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Table of Contents.

The Edward Stirling Lectures.[1]

LECTURE I: PRE-NATAL PÆDIATRICS.

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Thine eyes did see my substance, yet being imperfect:
and in thy book all my members were written;
Which day by day were fashioned: when as yet there was none of them.

—Psalm 139; version of "The Book of Common Prayer".

THE Charles Mickle Fellowship of the University of Toronto is awarded each decade to "that member of the medical profession who is considered to have done most during the preceding ten years to advance sound knowledge of a practical kind in medical art or science". Last month the University of Toronto awarded this Fellowship to Norman Gregg of Sydney in recognition of his dramatic incrimination of maternal rubella as a cause of certain congenital defects. It is with a special pride in Norman Gregg's brilliant discovery and its world-wide recognition and appreciation that I have chosen as my subject this evening that important aspect of preventive medicine which has been called "pre-natal pædiatrics", a subject which includes the study and possible control of those various "noxious influences" which may affect the normal growth and development of the embryo or the fœtus.

[1] Delivered on August 21 and 23, 1951.

For more than three thousand years the Chinese have officially acknowledged the fact that a newborn infant is about nine months old by awarding him a full year's credit on the day of his birth. This method of recording age represents a most appropriate recognition of the extremely important months which are spent in the uterus, for in the words of Sir Thomas Browne, " . . . we are all out of the computation of our age and everyman is some months older than he bethinks him; for we live, move, have a being and are subject to the actions of the elements and malice of diseases in that other world, the truest microcosm, the womb of our mother".

Various estimates have suggested that about 2% of all newborn infants exhibit some form of minor or major malformation, that the incidence of such defects is probably as high as 7% amongst premature infants, and that nearly 40% of dead fœtuses show some obvious malformation. Obviously these figures do not take into consideration all those congenital malformations which are not apparent at birth and may not become obvious until adult life or may never be recognized. Stockard offered a very sound assessment of this problem when he pointed out that congenital malformations were responsible for the death of more than 20% of the human race before or shortly after birth and handicapped a proportion of the survivors throughout their lives.

The falling infant mortality of recent decades has been responsible for a great increase in the relative significance of congenital malformations as a cause of death during the first year of life. In 1900 congenital malformations were apparently responsible for less than 3% of the infant mortality in Australia, but during the period from 1941 to 1945 these defects were regarded as being responsible for more than 12% of the infant mortality of this country

and as representing the second most common cause of death during the first month of life.

For many centuries the ætiology of congenital malformations was shrouded in mystery and superstition. During the Middle Ages gross congenital malformations were often interpreted as tokens of Heaven's displeasure or cited as examples of the craft and subtlety of the Devil or of "the foul fiend who gives the web and the pin, squints the eye and makes the hare lip; mildews the white wheat and hurts the poor creature of the earth" (Shakespeare, "King Lear", Act III, Scene 4). *Tractatus de Monstris*, a treatise written in 1570 by a French bishop, Arnaud Sorbin, included a series of excellent woodcuts illustrating certain gross malformations which the author ingeniously attributed to the wrath of Heaven provoked by events such as the arrival of Mohammed, the Anti-Christ, and the birth of Martin Luther (Figure I).

FIGURE I.

Reproduced from G. F. Still's "The History of Pædiatrics"; part of a page from Arnaud Sorbin's *Tractatus de Monstris* "showing the monstrosities due to the birth of Mohammed, the Anti-Christ".

The theory that impressions on the pregnant woman's imagination were responsible for the malformation of her infant was widely accepted during the sixteenth, seventeenth and eighteenth centuries, and yet Aristotle, whose knowledge of congenital malformations and their ætiology seems to have been far ahead of his time, denied the "maternal impression" theory and even suggested an appeal to comparative embryology. Hippocrates, who emphasized the fact that "generation . . . is not the same for the same seed in summer as in winter nor in rain as in drought", also denied this "maternal impression" theory. Despite his denial of this theory, Hippocrates is reported to have saved the honour of a fair princess, accused of adultery with a Negro, by pointing out that a large painting of a black slave on the wall of her bedchamber was responsible for the black pigmentation of her infant's skin—a story which may help to illustrate the Hippocratic sense of *noblesse oblige*.

The literature of the eighteenth century contains a number of fantastic reports which are offered as illustrations of the importance of maternal impressions as causes of congenital malformations. Among the best of these stories is that of an Englishwoman, Sarah Toft of Godalming, who was frightened by a rabbit while weeding a field and later gave birth to 17 rabbits; George I was intrigued by this report and directed his personal surgeon to investigate this remarkable delivery, which was vouched for and reported by the village surgeon, John Howard, in the year 1727. In the same year Boland published an essay on the power of the mother's imagination, and included a reference to a woman of Poland who "was brought to bed by two small fishes without scales which were no sooner born but they swam in the neighbouring waters naturally as others do". Sarah Gamp probably expressed the beliefs of her period when she spoke of a man "marked with a mad bull in Wellington boots upon his left arm, on account of his precious mother havin' been worried by one . . . when in a sitiwation which blessed is the man as has his quiver full of sech".

Early in the nineteenth century these "maternal impression" theories were discarded, the Mendelian laws were propounded and most congenital malformations were attributed to genetic defects. During the past decade an increasing knowledge of the possible ill effects of various maternal and external environmental factors on the developing embryo and fœtus has shaken our faith in the dogmatic generalization that most congenital malformations are genetically determined, and we now assume that the normal growth and development of a fertilized ovum depend firstly on its genetic constitution, over which we have no control except perhaps as eugenists, and secondly on various maternal and external environmental factors, some of which we may be able to control. Possibly some disturbances of development are the result of a combination of these two factors—a combination which Crew has aptly defined as "genetic inclination and environmental provocation"; in other words, it seems that a zygote may be particularly susceptible to certain changes in environment because of its genetic constitution.

Maternal Rubella.

Our present appreciation of the significance of maternal and external environmental factors in the ætiology of congenital malformations stems from Norman Gregg's discoveries. As most of you know, Gregg's original report, which emphasized such defects as cataracts, microphthalmos and congenital heart disease, appeared in 1941 and was followed by a detailed survey carried out by Swan and certain of his colleagues in this city, a survey which confirmed and amplified Gregg's original conclusions and defined further defects, including deaf mutism.

These discoveries opened up a new and significant aspect of "pre-natal pædiatrics" and focused medical attention on the first twelve weeks of intrauterine life, those hazardous weeks during which extremely rapid cell division is responsible for converting a single cell into a fœtus and for determining the ultimate structure of many important organs. As Ingalls has pointed out, the developmental changes of these first twelve weeks of intrauterine life are so rapid that a greater anatomical gulf exists between the organism and its predecessor of the previous week than is usually found between species, and it is probably during this so-called "organogenetic period" of intrauterine life that most congenital malformations are initiated.

The recognition of maternal rubella as a cause of congenital malformations helped to emphasize the significance of certain "critical periods" in the developmental life of organs and tissues, periods when very rapidly dividing cells are particularly susceptible to disturbance as a result of various "noxious influences". In contrast to these "critical periods" of the earlier weeks of pregnancy, Stockard has defined "moments of indifference", periods when quiescent primordia are much less susceptible to "noxious influences".

The available evidence suggests that the nature of the congenital defects following maternal rubella bears some direct relationship to the stage of pregnancy at which the mother developed rubella. Even when the date of the onset of the last menstrual period and the date of the appearance of the rash are known, any attempted estimation of the precise period of pregnancy at which the mother suffered from rubella must be far from accurate; but a review of 130 Australian case histories collected by Gregg in 1944 suggests that the incidence of cataracts was greatest when the rubella was stated to have occurred at about the fifth or sixth week of pregnancy, and that the incidence of deafness was greatest when the rubella was stated to have occurred at about the eighth or ninth week, times which are consistent with our theoretical concepts of the "critical periods" in the development of the lenticular and cochlear anlages (Figure II).

The diagrammatic representation of the "critical periods" in the development of the lens, cochlea and heart shown in Figure II may help to emphasize the embryological significance of the following additional observations based on the 130 case histories collected by Gregg: (i) that infants are rarely affected if the maternal rubella occurs after the fourteenth week of pregnancy; (ii) that eye defects are rare if the maternal rubella occurs after about the ninth week of pregnancy; (iii) that a combination of eye defects, deafness and congenital heart disease is rare.

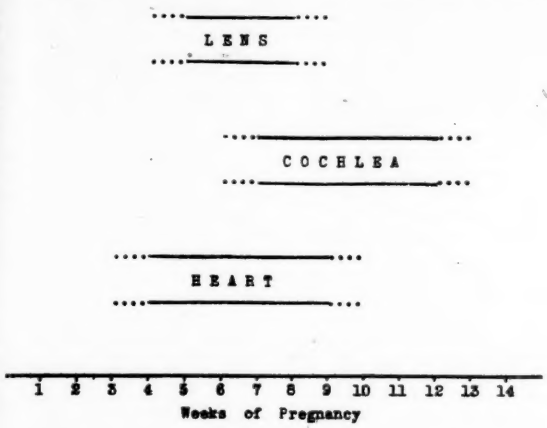

FIGURE II.
"Critical periods" in the development of the lens, cochlea and heart.

I do not intend to weary you with any further discussion of this maternal rubella problem, which has recently been reviewed in detail by Charles Swan of this city in his Bishop Harman Prize Essay; but before leaving this subject I should like to mention an Australian epidemic of deaf-mutism which occurred more than fifty years ago. Dr. H. O. Lancaster, lecturer in medical statistics at the School of Public Health and Tropical Medicine, Sydney, has drawn my attention to the fact that the Commonwealth Statistician emphasized in his 1933 report an "epidemic" of deaf-mutism amongst infants who were born at or about the turn of the last century, and argued that, as catastrophes such as war and famine had not occurred at this time, it was reasonable to suggest that this transient and significant increase in deaf-mutism might have been "the result of an extensive epidemic of infectious diseases which occurred at about the time that many of the affected children were born". Dr. Lancaster followed this clue by reviewing the dates of birth of all deaf persons admitted to the New South Wales Institution for the Deaf, Dumb and Blind, and established the fact that there was an "epidemic" wave of births of the deaf in 1899 (Figure III), which followed an epidemic of morbilli and rubella during the late winter and early spring of the previous year, 1898. I am grateful to Dr. Lancaster for allowing me to quote this evidence, which helps to illustrate an unfortunate hiatus between statistical and clinical medicine, for if the clinician had appreciated and interpreted these figures, maternal rubella as a cause of deafness might have been recognized at least thirty years earlier than it was. These figures also suggest that studies of the possible epidemicity of some of the commoner congenital malformations might be of some help in the elucidation of their ætiology.

Other Virus Diseases Occurring During Pregnancy.

The recognition of the fact that certain congenital defects followed maternal rubella naturally aroused interest in the possibility that other virus diseases occurring during pregnancy might be responsible for the production of congenital defects. During the past nine or ten years various malformations of the infant have been reported to follow such maternal diseases as measles, mumps, varicella, *herpes zoster*, infectious mononucleosis and so-called "influenza"; but there is not sufficient evidence to allow any statement to be made about the significance of these diseases as possible factors in the ætiology of congenital malformations.

The suggestion that vaccination against smallpox carried out during the early months of pregnancy might affect the development of the embryo has been denied by workers at the Sloane Hospital for Women, New York, who did

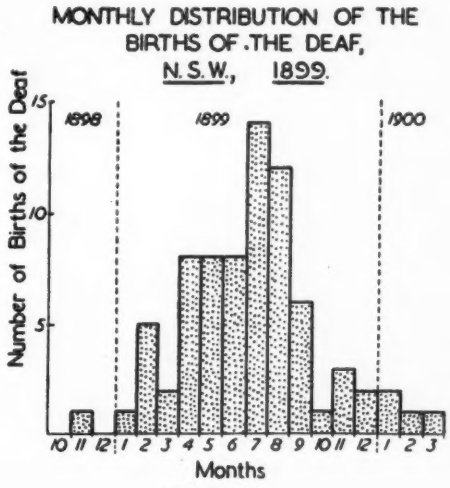

FIGURE III.

not find a significant increase in the incidence of congenital malformations amongst the infants of 633 mothers who were vaccinated during the early months of pregnancy.

Nutrition of the Mother.

The fœtus is usually regarded as a surprisingly successful parasite who achieves his nutritional requirements at the expense of his maternal host; but there is obviously a limit to this successful parasitism, and there is ample evidence of a direct relationship between the mother's nutrition during her pregnancy and the physical status of her newborn infant. During 1945, in the city of Rotterdam, Clement Smith studied a large group of infants born to mothers who had suffered six months or more of hunger and semi-starvation during their pregnancies, and these studies established a significant diminution in birth weight and length and in increased incidence of still births.

Warkany's brilliant demonstration that various congenital malformations including hare-lip, cleft palate, short limbs and short mandibles may be induced by depriving the maternal rat of riboflavin during certain periods of her pregnancy, and his demonstration that other congenital malformations of the newborn rat such as eye defects, abnormalities of the renal tracts and anomalies of the heart and great vessels may follow vitamin A deprivation during pregnancy, are probably well known to many of you. Duraiswami has shown that the injection of insulin into the yolks of developing hen's eggs may give rise to a number of developmental abnormalities in the chickens, including *spina bifida*, malformations of the legs, eyes and beaks, and certain skeletal defects resembling *osteogenesis imperfecta*. This worker maintains that the affected embryo develops hypoglycæmia which persists for several days, and has suggested that this is responsible for damage to the rapidly developing embryonic cells, which apparently depend for their nutrition on the metabolism of glucose.

These and other animal experiments have stimulated interest in the possibility that nutritional deprivation during the early months of pregnancy may be responsible for the congenital malformation of the infant, but as far as I know a direct relationship between the mother's nutrition

during her pregnancy and the congenital malformation of her infant has never been established. As Warkany has pointed out, such a nutritional deficiency would need to be "border-line" in degree, as a moderate defect in the mother's diet would be unlikely to disturb the development of the infant, while a gross deficiency might result in death of the fœtus. I feel that a carefully controlled "prospective" study of the live and the stillborn infants of mothers who had suffered from excessive vomiting during the early months of their pregnancies might throw some light on this question.

Age of the Mother.

It seems that there is a significant increase in the incidence of congenital malformations amongst infants born to mothers of more than thirty-five years of age, and that the incidence of these defects amongst infants born to mothers of more than forty years is probably three times as great as the incidence amongst the infants of mothers of less than thirty years. It has been estimated that if the average age of the pregnant mother could be reduced by five years, the number of infants suffering from congenital malformations would be halved.

Age of the Father.

There seem to be no published studies of the incidence of congenital defects among infants born to the young wives of old husbands, and I am sure that you will agree that various human frailties would make this a dangerous field for statistical research.

Uterine Hæmorrhage Occurring During the First Three Months of Pregnancy.

Various authorities have emphasized the association of significant uterine hæmorrhage occurring during the first trimester of pregnancy and the birth of a malformed infant who is presumably the lucky or the unlucky survivor of a threatened miscarriage. Dr. F. N. Street of Sydney has drawn my attention to the fact that uterine hæmorrhage occurring during the first three months of pregnancy is occasionally followed by the birth of a mature but miniature infant without any obvious congenital malformation.

Hydramnios.

Many authorities have stressed the association between hydramnios and congenital defects. It has been estimated that approximately 10% of the mothers of normal infants, 25% of the mothers of infants born with tracheo-œsophageal fistulæ and 51% of the mothers of anencephalic monsters, suffer from hydramnios. Whatever the pathological process responsible for hydramnios may be, this frequent association with gross congenital malformations suggests that the original pathological changes probably occur during the first trimester of intrauterine life.

Properly controlled "prospective" studies of the live and stillborn infants of mothers who have suffered from uterine hæmorrhage or hydramnios may help to establish the possible significance of such disturbances as causes of congenital malformations.

Abortifacients and Spermaticides.

Very little is known about the effect of sublethal doses of abortifacient drugs on the developing embryo or fœtus, but it seems reasonable to assume that these drugs, which are usually taken during the important "organogenetic" period of intrauterine life, may be responsible for a certain number of congenital malformations. It is even remotely possible that certain spermaticide drugs may by disturbing the vitality of the spermatozoon affect the ultimate development of the zygote.

Prolonged Periods of Infertility.

Murphy and others have emphasized the increased incidence of congenital malformations after "prolonged periods of infertility", and figures have been produced to support this hypothesis; but much to my surprise the incidence of congenital malformations amongst infants born to mothers attending sterility clinics in Sydney has been extremely low. Dr. Alan Grant informs me that of 232 infants born to mothers attending the sterility clinic of the Women's Hospital, Crown Street, Sydney, during the period from 1945 to 1950, only three showed obvious congenital malformations at birth, and Dr. J. W. Knox found no obvious congenital malformations amongst 45 infants born to mothers attending the sterility clinic at the King George V Memorial Hospital for Mothers and Babies, Sydney, during the period 1949-1950.

Other Noxious Influences Occurring During Pregnancy.

Radium or X-ray treatment of the pelvis during early pregnancy represents a rare but well-established cause of congenital malformations of the infant, microcephaly and mental retardation being by far the most common defects produced.

It seems that a small number of congenital malformations may be the result of intrauterine posture or pressure, but I think that this group of deformities is much smaller than Denis Browne and others have suggested.

Gross congenital malformations are rarely associated with congenital syphilis, probably because the spirochæte does not reach the fœtus until some time after the first four months of intrauterine life.

Similarly hæmolytic disease of the fœtus and the newborn is rarely associated with gross congenital malformations of the infant, as the fœtus is not affected by this disorder until after the "organogenetic" period of development has been completed. The ætiological mechanism responsible for hæmolytic disease of the fœtus and the newborn represents an excellent example of an overlapping of genetic and environmental factors—a genetic factor responsible for the blood group of the fœtus and a maternal or environmental factor represented by the iso-immunization of the mother and the consequent injury of the fœtus.

Mongolism.

Although it has been argued that genetic factors may occasionally play some part in the ætiology of mongolism, the bulk of the available evidence suggests that maternal and external environmental factors acting during the "organogenetic" period of the embryo's life are responsible for the multiple developmental "arrests" and anomalies which constitute this syndrome.

The various physical abnormalities which may be associated with mongolism, including anomalies of the little fingers, the nasal bones, the palate, the external auricles, the basilar portion of the skull, the heart and the lens of the eye are probably initiated between the sixth and ninth weeks of intrauterine life. Most embryologists would agree that during these three or four weeks of the intrauterine life of the normal infant the cardiac septa are forming, torsion of the great vessels is occurring, the lens of the eye is undergoing very rapid development, the secondary lens fibres are appearing, fusion of the tubercles of the external auricles is occurring, the nasal bones, the palate and the maxillæ are beginning their development, and the middle phalanges of the fingers are appearing, the middle phalanx of the little finger, which may be absent or unduly short in the mongol, being the last to evolve. Apparently the cataracts occasionally found in the eyes of mongoloid children are usually arcuate opacities which are probably initiated during the eighth week of intrauterine life when the early secondary lens fibres are appearing.

Unfortunately, we cannot define the noxious influences which may be responsible for these developmental defects. We know that mongols are frequently born to mothers in the later age groups, but we also know that a young mother's first-born child may be a mongol.

Like many other observers, I have been impressed by the frequency with which a uterine hæmorrhage during the first trimester of pregnancy may be followed by the birth of a mongol; but although advanced age of the mother and uterine hæmorrhage occurring during the first trimester of pregnancy appear to be the most important known factors in the ætiology of mongolism, it is possible

that other "noxious influences" affecting the embryo during the early weeks of pregnancy may also play some part in the ætiology of this syndrome; in other words, the mongol may be regarded as the unfortunate survivor of a disturbance occurring at about the second month of pregnancy, and any one of a number of factors may be responsible for this disturbance.

Maternal virus infections occurring during the early weeks of pregnancy are very rarely followed by the birth of a mongol, but the following case history may be of interest in this respect:

An apparently healthy young mother, aged twenty-two years, developed mumps at about the fourth to sixth week of her first pregnancy. While she was suffering from this illness her two nieces, who were living in her house, developed rubella, and about ten days later she developed a fever which persisted for approximately four days. This mother's infant unfortunately exhibited all the classical features of mongolism and also many other congenital malformations, including absence of the middle phalanx of the right little finger, a "lobster claw" deformity of the left hand, absence of the toes of the left foot, a hare-lip and cleft palate, bilateral central cataracts and microphthalmos, and a cyanotic congenital cardiac lesion. A healthy and vigorous male child born to the same mother about a year later did not present any obvious congenital abnormalities.

In discussing the question of future children with the mother of a mongol, we should be aware of the fact that more than one mongol may be born to the same mother, and that the risk of mongolism occurring amongst the subsequent infants of a mother who has already borne a mongol is significantly greater than if no mongoloid child had been born in the family. Although Brenda in a review of 255 mongols found only two incidences of multiple mongolism occurring in a family, this unfortunate occurrence is probably more common than these figures would suggest. As some evidence in support of this statement, I know of three families in which more than one child was a mongol, and two of these families were referred to me by Dr. Donald Vickery during the past six months.

Other Congenital Malformations of the Central Nervous System.

The ætiology of many gross congenital malformations of the central nervous system, such as hydrocephalus, microcephalus and *spina bifida*, is confused by an overlap of genetic and environmental factors; but it seems that environmental factors may be responsible for a number of these defects. As a result of a study of 1729 mentally defective children, Yannet has suggested that about 40% of the mental deficiency of childhood may be the result of ætiological factors operating during the pre-natal period. Dorothy Russell has concluded that various developmental errors, and more particularly "forking" of the aqueduct of Sylvius, are often responsible for congenital hydrocephalus, and has emphasized the frequent association of these defects with varying grades of *spina bifida* and their occasional association with mid-brain malformations, the Arnold-Chiari syndrome and other congenital malformations of the central nervous system. Is it possible that some of these defects are the result of various noxious influences occurring during the early weeks of pregnancy?

Congenital Cardiac Defects.

Although genetic factors may play a part in the ætiology of congenital cardiac defects, it seems probable that many congenital cardiac defects are the result of some maternal or external environmental factor operating during the early weeks of pre-natal life.

Wilson and Warkany have recorded a series of cardiac defects and aortic arch anomalies in the offspring of vitamin *A* deficient rats; these congenital cardiac malformations were associated with abnormalities of the eyes, genito-urinary system and diaphragm, and some of the rats exhibited vertebral defects similar to those occasionally associated with aortic arch and other cardiac malformations in the child. We know that congenital heart disease may follow maternal rubella occurring somewhere between the fifth and ninth weeks of intrauterine life, that it is often associated with mongolism, which we assume to be the result of some "noxious influence" acting during approximately the same period of pregnancy, and that there is possibly some evidence to suggest that uterine hæmorrhages occurring during the early weeks of pregnancy may occasionally be responsible for congenital malformation of the heart.

Occasionally there may be a history of congenital cardiac disease amongst other members of the family. Abbott and Brown have recorded a total of 17 pairs of brothers and sisters suffering from congenital heart disease, and Taussig has reported four families each containing more than one affected member. I am aware of two families in each of which two children suffered from patency of the *ductus arteriosus*, and of a third family in which all three children suffered from a congenital cardiac defect. This third family was brought to my notice by Mr. J. Steigrad, who kindly allowed me to examine an infant suffering from a congenital cardiac defect and polydactylism.

The mother of this child was born with a supernumerary finger on each hand and presented clinical evidence suggesting a possible intraventricular septal defect; her first child had six fingers on each hand and died of cyanotic congenital heart disease at the age of four years; her second child had supernumerary fingers and toes and died of cyanotic congenital heart disease (*truncus arteriosus* and patency of the intraventricular septum) at the age of four months; her third child has a supernumerary finger on each hand and suffers from a congenital cardiac defect which has been classified as an intraventricular septal defect (Figure IV).

I have reviewed the pre-natal histories of 162 children who have attended the Congenital Heart Clinic of the Royal Alexandra Hospital for Children during the past three years; two children in this series were suffering from congenital heart defects which were apparently genetically determined, and in 34 instances there was a history of some "noxious influence" during the early months of pregnancy (rubella 22, other "noxious influences" 12). A recent clinical review of children suffering from congenital malformations following maternal rubella, which was carried out in Sydney during January, 1949, was probably responsible for the surprisingly high incidence of post-rubella congenital cardiac defects in this series. Apparently the most common congenital cardiac defect to affect more than one member of a family is patency of the *ductus arteriosus*, and this lesion also seems to be the most common congenital cardiac defect to follow maternal rubella. Jackson and others have emphasized the fact that cyanotic congenital heart disease is a very rare and patency of the *ductus arteriosus* a very common type of lesion to develop as a result of maternal rubella during the early weeks of pregnancy. Jackson asks why a maternal infection of this type occurring six or more months before birth should delay or prevent closure of the *ductus arteriosus* during post-natal life, and wisely concludes that as we do not know how the ductus closes in the normal infant it would be idle to speculate about disturbances of this mechanism.

Most of the literature about congenital cardiac malformations associated with mongolism suggests that *atrioventricularis communis* is by far the most common defect found in these children; but a recent review by Evans of the post-mortem records of 28 mongols suffering from congenital cardiac defects has established the fact that ventricular and auricular septal defects were by far the most common lesions, accounting for more than two-thirds of the cardiac defects, and that *atrioventricularis communis* was found in only four of the 28 infants.

Why is cyanotic congenital heart disease relatively rare amongst mongols and very rare as a sequel of maternal rubella? Why should patency of the *ductus arteriosus*, which is relatively rare amongst mongols, be by far the most common cardiac defect to follow maternal rubella? The study of these and associated problems by the embryologist and the cardiologist might teach us something about the ætiology of congenital cardiac defects.

Maternal Diabetes.

Numerous authorities have stressed the high incidence of congenital defects amongst the infants of diabetic mothers, defects which have occurred despite adequate

control of the mother's diabetes and various forms of hormonal therapy during pregnancy. It seems that even though we may be able to control the excessive weight and length, the advanced osseous age and other classical abnormalities of the newborn infants of diabetic mothers, the increased incidence of congenital malformations amongst these infants is beyond our control at present.

Sheumack studied 52 infants born to diabetic mothers in the Royal Hospital for Women, Sydney, during the years 1935-1950, and found only two infants suffering from obvious congenital malformations. But Priscilla White has recently informed me that the incidence of gross and obvious congenital malformations amongst more than 400 infants born to diabetic mothers under her care was in

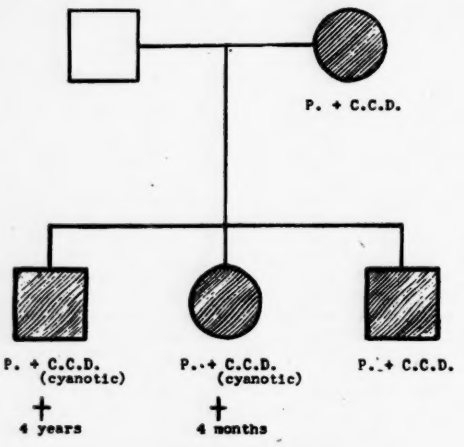

FIGURE IV.
Polydactylism and congenital cardiac defects.

the neighbourhood of 10%, and that these gross congenital malformations included "congenital heart disease of many types, syndactyly, claw hand, meningocele, cleft palate and hare-lip, mermaid deformity, absence of kidneys and ureters and absence of gonads".

How are we to explain this very high incidence of gross congenital malformations, most of which were probably initiated during the "organogenetic period" of intrauterine life? Is it possible that the pituitary growth factor, which we assume plays an important part in the ætiology of diabetes, and which is probably responsible for the excessive growth and rapid epiphyseal maturation of the newborn infant of the diabetic mother, may increase the risk of congenital malformation by hastening cell division and growth during the organogenetic period of intrauterine life?

Retrolental Fibroplasia.

The label of retrolental fibroplasia is not a very satisfactory one and lacks accurate definition, but for those of us who are not ophthalmologists this condition may be reasonably defined as a form of retinal detachment which usually becomes apparent during the early weeks of the extrauterine life of a premature infant, and which is followed by the formation of a greyish-white opaque membrane behind the lens; most of the affected infants suffer from blindness of a severe degree, and microphthalmos is usually obvious.

During the past decade this condition has been recognized with increasing frequency in certain countries, and more particularly in the United States of America, where it has recently been incriminated by Reese as a cause of approximately one-third of all blindness amongst pre-school children. Apparently the incidence of this condition varies widely in different parts of the world, in different cities in the same continent and even in different hospitals in the same city. A review of premature infants born in the Boston Lying-in Hospital between 1940 and 1945 established the fact that approximately 10% were affected and that the incidence was as high as 16% amongst those infants who weighed less than three pounds at birth, while a survey of 4000 premature infants born in Birmingham revealed an overall incidence of only 0·1% and an incidence of about 4% amongst infants weighing less than three pounds at birth.

Early this year Dr. Eben Hipsley reviewed 86 premature infants who were born in Sydney during the five-year period from 1945 to 1949 and who weighed less than three pounds at birth; Dr. Norman Gregg found that five of these 86 infants presented the clinical features of retrolental fibroplasia. None of the five affected infants presented any obvious evidence of other congenital malformations at the time of examination, and three of them had weighed less than two pounds at birth.

Although retrolental fibroplasia may develop in the eyes of a full-term infant, it is much more frequent amongst premature infants, and its incidence seems to be inversely proportional to the birth weight. Clinical evidence of this defect is rarely, if ever, apparent at birth, the first signs do not usually appear until the infant is more than four weeks old, and the process seems to reach a "quiescent" stage at or before the age of six months.

Is this defect pre-natal or post-natal in origin? Or is it possible that the original disturbance occurs during the pre-natal period, and that the other progressive changes observed during the early weeks of life are the result of some post-natal factor or factors?

There seem to be two distinct schools of thought about this problem. One school emphasizes the association with other congenital malformations and the occasional presence of this defect in the full-term infant, and argues that the condition is probably determined during pre-natal life.

The other school, which seems to have the weight of evidence on its side, denies the association of retrolental fibroplasia with other congenital malformations, emphasizes the obvious association with prematurity and the fact that the clinical signs do not begin to develop until some weeks after birth, and makes the assumption that the defect is essentially post-natal in origin. This argument, which has a great deal to support it, has prompted various attempts to incriminate certain factors operating during the early weeks of the premature infant's extrauterine life, and Dr. Kate Campbell's recent suggestion that prolonged and generous administration of oxygen during the early weeks of life may play an important part in the ætiology of this condition prompts a wide-scale investigation in this and other countries.

At present there is no proven solution to this problem of retrolental fibroplasia; but whatever the final solution may be, the ophthalmologist who observes this condition in the eyes of a small premature infant is watching with his own eye the day-to-day and week-to-week progression of an intrauterine developmental anomaly, for the premature infant of low birth weight is essentially a fœtus for several weeks, even though he has left the uterus and is living in the outer world.

Toxoplasmosis.

The natural mode of transmission of the protozoan parasite toxoplasma is not known, but apparently very mild or symptomless maternal toxoplasmosis may be responsible for a severe infection of the fœtus, whose nervous system bears the brunt of the disease.

Toxoplasmosis is probably commoner in this country than we realize. Graeme Robertson of Melbourne has published details of three cases and informs me that he has seen two other patients who are probably suffering from this disease; Edmonds has reported a case from Western Australia; and I am aware of four cases recognized in Sydney, one of which was confirmed by post-mortem examination.

The classical clinical features of this condition include hydrocephalus or microcephalus, microphthalmos, and bilateral focal chorio-retinitis involving the macular region. Flecks or curvilinear streaks of intracranial calcification are usually evident in radiographs of the skull. During the early and active phases of the disease the cerebro-spinal fluid is usually xanthochromic, the protein level significantly elevated and the number of mononuclear cells increased.

The following history of an infant suffering from toxoplasmosis illustrates some of the classical features of this condition:

A male infant was admitted to the Royal Alexandra Hospital for Children on the third day of life because of fever and repeated convulsive movements, and died within three days of admission. An X-ray examination of his skull showed marked widening of the sutures and some "flecks" suggesting intracranial calcification. The cerebro-spinal fluid was xanthochromic, contained 40 mononuclear cells per cubic millimetre, and had a protein content of more than 1000 milligrammes *per centum*. The diagnosis of toxoplasmosis was made by Dr. Douglas Reye at the post-mortem examination and was confirmed serologically by Dr. Feldman, of Syracuse, United States of America.

The developmental anomalies such as the microcephalus and the microphthalmos which may be associated with toxoplasmosis are probably the result of disturbances of the later stages of development of the central nervous system caused by this pre-natal inflammatory process. And so it seems that a simple anatomical defect such as microphthalmos may be the result of maternal rubella occurring during the early organogenetic phase of embryonic development, or of an intrauterine infection such as toxoplasmosis possibly occurring at a later stage of fœtal development, or associated with retrolental fibroplasia, or may be genetically determined. In other words, the ætiology of microphthalmos represents an excellent example of the old adage that "what heredity can do, environment can also do".

Conclusion.

I know that many congenital malformations are genetically determined, but I am anxious to stress our responsibility in relation to that large group of congenital malformations which are not genetic in origin. Gregg and other workers have pointed the way, and it is now our responsibility to seek out those maternal and external environmental factors which may be responsible for the malformation of the infant, to do what we can to protect pregnant mothers from these hazards and so to prevent at least some of these congenital malformations which throw their shadows over many homes at the present time.

Bibliography.

Benda, C. E. (1946), "Mongolism and Cretinism: A Study of the Clinical Manifestations and the General Pathology of Pituitary and Thyroid Deficiency", New York, Grune and Stratton, Incorporated.

Brown, J. W. (1938), "Congenital Heart Disease", John Bale Medical Publications, London.

Browne, Denis (1949), "Congenital Deformities", in Garrod, Batten and Thursfield's "Diseases of Children", Volume I, page 17.

Campbell, K. (1951), "Intensive Oxygen Therapy as a Possible Cause of Retrolental Fibroplasia", THE MEDICAL JOURNAL OF AUSTRALIA, Volume II, page 48.

Crew, F. A. E. (1947), "Genetics in Relation to Clinical Medicine", Oliver and Boyd, Edinburgh.

Gregg, N. M. (1941), "Congenital Cataract following German Measles", *Transactions of the Ophthalmological Society of Australia (British Medical Association)*, Volume III, page 35.

Ingalls, T. H. (1947), "Pathogenesis of Mongolism", *American Journal of Diseases of Children*, Volume LXXIII, page 279.

Jackson, A. V. (1950), "Congenital Heart Disease and Rubella", page 255, in "Studies in Pathology: Presented to Peter MacCallum", edited by E. S. J. King, T. E. Lowe and L. B. Cox, Melbourne University Press, page 255.

Robertson, E. G. (1946), "Toxoplasmic Encephalomyelitis, with the Report of Two Cases", THE MEDICAL JOURNAL OF AUSTRALIA, Volume II, page 449.

Robertson, E. G., and Hamilton J. B. (1945), "Toxoplasmic Encephalomyelitis and Chorioretinitis", *Transactions of the Ophthalmological Society of Australia (British Medical Association)*, Volume V, page 85.

Russell, D. S. (1948), "Observations on the Pathology of Hydrocephalus", Medical Research Council of the Privy Council, Special Report Series Number 265, page 14.

Still, G. F. (1931), "History of Pædiatrics", Oxford University Press.

Swan, C., Tostevin, A. L., Moore, B., Mayo, H., and Black, G. H. B. (1943), "Congenital Defects in Infants following Infectious Disease during Pregnancy", THE MEDICAL JOURNAL OF AUSTRALIA, Volume II, page 201.

Swan, C., Tostevin, A. L., Mayo, H., and Black, G. H. B. (1944), "Further Observations on Congenital Defects in Infants following Infectious Disease during Pregnancy, with Special Reference to Rubella", THE MEDICAL JOURNAL OF AUSTRALIA, Volume I, page 409.

Swan, C. (1949), "Rubella in Pregnancy as an Ætiological Factor in Congenital Malformation, Stillbirth, Miscarriage and Abortion", *The Journal of Obstetrics and Gynæcology of the British Empire*, Volume LVI, pages 341 and 591.

Taussig, H. B. (1947), "Congenital Malformations of the Heart", Commonwealth Fund, New York.

Warkany, J., and Schraffengerger, E. (1944), "Congenital Malformations Induced in Rats by Maternal Nutritional Deficiency", *Journal of Nutrition*, Volume XXVII, page 477.

Warkany, J. (1947), "Ætiology of Congenital Malformations", in "Advances in Pediatrics", Volume II, page 1, Interscience Publishers, Incorporated, New York.

Yannet, H. (1950), "Mental Deficiency due to Prenatally Determined Factors", *Pediatrics*, Volume V, page 238.

CORTICAL NEUROLOGY: VISUAL DISTURBANCES AND THE OPTIC PART OF THE BODY IMAGE.

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PART I.

THE following brief historical survey traces some of the important contributions to our understanding of the relationships between structure and function in the realm of vision. A key to much of the earlier work is provided by Fulton (1949).

That vision depends upon the integrity of the cerebral cortex was first established experimentally by Fleurens in 1823, but it was then believed that ablation of one hemisphere caused blindness of the opposite eye. This belief was confirmed by Bouillard, Magendie, Longet and others; it was noted that the pupil still reacted to light. Panizza in 1855 localized vision in the posterior part of the cerebrum, clinical cases indicating the parieto-occipital cortex, but experiments on dogs narrowing the visual area to the occiput. Hitzig in 1874, apparently independently, performed similar experiments on dogs. Schafer showed that in monkeys the occipital lobes contained the primary visual cortex, and that ablation of one lobe led to contralateral homonymous hemianopia, not to monocular blindness. The work of Schafer in animals, and of Henschen and Wilbrand, narrowed the localization to the *area striata*. Experimental proof came from Minkowski working on dogs, from Poljak (1933) working on monkeys, and from Holmes (1918*a*) in studies on man. Minkowski in 1917 showed that lesions in the cortex other than in area 17 did not cause significant visual disturbance, and the anatomical evidence of the visual projection from the lateral geniculate body to the *area striata* followed. The functions of area 18 (the intra-occipital associations concerned with the organization of the visual image) were discussed by Holmes (1918*b*). Those of area 19 (the visual associations involving other sensory and motor areas) were studied by Horrax (1923).

Visual Associations.

Visual disorganizations due to lesions of visual association areas were described by Riddoch (1917 and 1935), and by Horrax, Holmes (1919) and Brain (1941). Visual hallucinations of highly organized episodes have been described by Cushing and by Horrax and Putnam (1932) as due to tumours compressing area 19. Sachs (1931), Bailey (1933) and Kinnier Wilson (1940) stressed the importance of unformed hallucinations (usually coloured) in the diagnosis of occipital lobe tumour. Formed visual elements usually indicate spread to the temporal lobe. These authors and Gowers (1893) described the typical features of such occipital hallucinations. However, Cushing

and Bailey (1928) describe two cases of aneurysmal varix overlying the occipital cortex, in which, in spite of papillœdema and optic atrophy in one, and papillœdema and partial homonymous hemianopia in the other, no visual hallucinations occurred, in contrast to cases of tumour in the substance of the occipital lobe.

Bonin, Garol and McCulloch (1942) explored the physiological organization and interrelations of areas 17, 18 and 19 by analysing cortical electrograms following local strychninization.

The Parieto-Temporo-Preoccipital Cortical Unit.

Psychic blindness was described by Kluver and Bucy (1938, 1939) and further elucidated by the studies of Bailey *et alii* (1943*a*). These studies showed that the whole ventro-lateral surface of the temporal lobe projected to area 19, and was thus a visual association area. Ades and Raab (1949) demonstrated that simple visual form discrimination was abolished by one-stage bilateral extirpation of areas 18 and 19, but that the discrimination between a black and a white card was not. However, retraining reestablished the discrimination habit just as easily as the initial training. Bitemporal cortical ablation did not abolish the habit or delay initial training. Bilateral ablation of one region, followed after an interval by bilateral ablation of the other, in either order, permanently abolished this simple visual form discrimination. With training trials between stages, a two-stage bilateral extirpation of areas 18 and 19 did not disturb the habit. These studies confirm that the temporal cortex is a visual association area.

Blum, Chow and Pribram (1950) showed that form and colour discrimination was disturbed by temporal lobe ablations. These authors (1950) made a behavioural analysis of the organization of the parieto-temporo-preoccipital cortex. In this paper they corrected the common error of attributing undue importance to the frontal granular cortex as "the organ of civilization". They quote the points made by von Bonin (1941) that there is no significant difference in the relative size of the frontal lobes between man and the chimpanzee, and that man's mental superiority may be partly explained by the relative increase of the parietal and temporal lobes, the areas in which lesions cause aphasia. They also point out that if anything distinguishes man from phylogenetically lower primates it is the development of propositional language. Of interest here is the study by Turner (1950), who traced the ontogenetic development of the whole cerebral cortex and of each lobe. He found that the parietal lobe enlarges by five times from birth to the second year, the frontal lobe and the whole cortex each by four, the occipital lobe by three to four, and the temporal lobe by a little less. The experiments on monkeys reported in this contribution by Blum, Chow and Pribram (1950) were based on the hypothesis that the granular cortex of the parieto-temporo-preoccipital areas forms one functional unit, like that of the prefrontal lobes. This explained why parietal, temporal or preoccipital ablations had not singly produced highly significant dysfunctions. These workers showed that, while parietal lesions caused somatæsthetic disturbances, and preoccipital lesions caused visual disturbances, parieto-temporo-preoccipital ablations caused much more significant disturbances in the more complex somatæsthetic and visual functions. Moreover, while temporal lobectomy alone produced little effect upon somatæsthetic discrimination, it produced disturbances in visual functions similar to those produced by parieto-temporo-preoccipital ablation. This was confirmed by Chow (1950*a*), who produced similar visual defects by lesions within the lateral surface of the temporal neocortex. This importance of the posterior temporal cortex reminds us again of Head's (1923, 1926) concepts of semantic, syntactic and nominal aphasia, defects caused by lesions in the supramarginal gyrus, the superior temporal gyrus and the angular gyrus respectively, in the dominant hemisphere. The speech functions concerned involved complex organization and meaningful patterns in thought processes concerned with speech. Also, in this posterior temporal area, we find lesions causing disturbances of the body-image, without actual somatæsthetic or visual sensory loss (Schilder, 1935). Again, lesion in the supramarginal gyrus on this side may produce bilateral astereognosis, as stated by Spiegel and Sommer (1944), and asymbolia for pain and danger, as reported by Schilder and Stengel (1931).

In this parieto-temporal area, as Ritchie Russell (1948) points out, established patterns of function of a highly organized and intellectual kind are located, or it is at this site that these functions are critically susceptible to disturbance by circumscribed lesions. Le Gros Clark (1948), too, draws attention to the greater relative phylogenetic development of the parieto-temporal lobe. It appears to be the zone of vital importance for the recognition and utilization of *Gestalten* of all kinds.

Chow (1950*b*) has shown by retrograde cell degeneration studies much new detail of the cortical projection field of the pulvinar to this area. His contribution gains enormously when read in conjunction with the above-mentioned papers on localization, and especially with another paper by Schilder (1934).

Phylogeny.

Fulton (1949), in reviewing the phylogeny of visual function, refers to the work of Marquis (1934, 1935), which traces the progressive encephalization of visual function from the tectum in fish and amphibia to the occipital cortex in man. The primitive visual reflexes remain centred in the tectum. In the monkey, practically no serviceable vision remains after double occipital ablation (Kluver, 1936); but a primitive light discrimination of a crude type can be demonstrated by sensitive conditioned reflex techniques. These techniques demonstrate that the animal is aware of shadows passing across the visual field (Marquis and Hilgard, 1936).

Orientation in Visual Space.

Thus, it is the elaborate spatial organization of the geniculo-calcarine projection, and areas 17, 18 and 19 and the ventrolateral temporal cortex, that makes object vision, spatial perception and orientation in visual space possible.

Visual Function in Perimetrically Blind Fields.

Marquis (1934) stated that man was completely and permanently blind after bilateral occipital ablation; but M. B. Bender and his various co-workers have published an extensive series of articles on their investigations of occipital lobe injury in World War II casualties. Bender and Krieger (1951) demonstrated that some visual function remained "in perimetrically blind fields" (fields of vision that were mapped as scotomata or blind fields in standard perimetry) when the subjects were tested in total darkness with a red pin-point light as a fixation point and a small light as the perimeter target. Below a certain light intensity of target only "glow vision", a diffuse illumination like the sky of the piccaninny dawn, was found; but as soon as a certain threshold intensity of light was reached a blurred "light was seen superimposed on the glow". Among the characteristics of this function discussed were the ability of such subjects to localize, manually or verbally, the "light superimposed upon the glow", and subjective experiences reported by these subjects that revealed that they had already noted these phenomena for themselves. Such a case is presented in this paper.

Cerebellar Projections.

A review of recent contributions to cerebellar anatomy and physiology by Ray Snider (1950) contains information that suggests a possible physiological basis for the above-described phenomena, despite the destruction of geniculo-calcarine projection fibres. Snider and Stowell (1942, 1944*a* and *b*) claimed to have demonstrated in the cat a tactile projection from gracile and cuneate nuclei via arcuate fibres to the cerebellar cortex. The work of Dow and Anderson (1942) suggests that the cerebellar areas for tactile and proprioceptive projections are coextensive. This raises again the view of von Frey, as quoted by Schilder

(1935), that there are no deep sensations coming from the joints, and that only the special configuration of stimuli on the skin leads us to the perception of passive movements of our limbs. Stowell and Snider (1942*a*) similarly demonstrated an auditory projection, and again (1942*b*) a visual projection coextensive with the auditory area of the cerebellar cortex—an audio-visual area. This projection is mediated via the superior colliculus (Snider, 1945). These contributions, and the work of Sjoqvist and Weinstein (1942), and of Snider and Barnard (1949), in particular, are collated in the review by Snider (1950) to support the view that there is a double projection of tactile, auditory and visual functions (the well-known paths to the cerebral cortex, and these newly discovered paths to the cerebellar cortex), and to show that there is a two-way connexion between the cerebral and the cerebellar cortical area for each of these senses. Therefore, Snider points out, each may modulate, positively or negatively, the function of the other, in an elaborate feed-back mechanism. A similar suggestion was put forward by Lettvin of Manteno State Mental Hospital, Illinois, during the clinical discussion on a patient who had suffered a pontine lesion. The medial lemniscus and the seventh and probably the fifth cranial nerve were destroyed. Skin stimuli still gave rise to sensory responses; but they were diminished and delayed. Lettvin's suggestion was that this delay corresponds with the delay consequent upon the spino-cuneato-cerebello-cerebral bypass (W. R. Adey, personal communication). Possibly such a bypass subserves the visual function in "perimetrically blind fields". The man reported upon in this study, with such visual function in his right hemianopic fields, also showed delayed response to somatæsthetic stimuli in the right lower limb.

The Work of Bender and his Co-Workers.

The fruitful application to neurology of *Gestalt* principles of Wertheimer, Koffka and Kohler, notably by Goldstein, Gelb, Kluver and others, is reflected in these later references, and in papers of M. B. Bender and his co-workers of Bellevue, New York. From a comprehensive series of studies, some may be taken as of special interest here. Bender *et alii* studied, in patients with occipital and parieto-occipital lesions and diffuse lesions also involving these areas, the disturbances of somatæsthetic, visual, speech and body-image functions.

Physiological Basis for Body-Image Disturbance.

Bender and Furlow (1945) discussed the question of visual extinction in impaired but perimetrically intact fields in a man with left parieto-occipital damage with initial right homonymous hemianopia, severe acalculia, spelling defect and dysgraphia. As the hemianopia cleared, residual visual extinction phenomena became apparent and were explained on the Goldstein (1944) theory of lability of threshold in a damaged cortex—an explanation in terms of rivalry, dominance and attention mechanisms.

In a report dealing with changes in sensory adaptation time and after-sensation in two of his patients with parietal lobe lesion, Bender (1946) refers again to rivalry with resultant dominance in visual and tactile spheres, and demolishes Poppelreuter's theory that visual extinction is due to "inattention", by citing the conditions under which it occurs. Critchley (1951) has attempted to explain body-image disturbance by "inattention" also, and is critical of Bender's use of the term extinction. However, one must remember the special sense in which Head (1923) and presumably Critchley (who refers to their papers) used this term, "attention" being a function of the parietal cortex endowing the capacity to select, integrate and act upon patterns of sensory stimuli. Nevertheless, such an explanation is now inadequate to meet the facts, particularly the results of simultaneous symmetrical stimulation in such cases as Bender's (1946), the first of which closely resembles the one reported in the present study. Ritchie Russell (1951), in the same discussion as Critchley (1951), uses Bender's terms and suggests an explanation for extinction. When one parietal cortex is damaged, the ipsilateral cortex provides for the residual function of the affected side of the body. When the normal side of the body is stimulated, these stimuli are prepotent in activating the undamaged cortex, so that the stimuli from the affected side suffer extinction. It is also suggested that the relay of the stimuli to the ipsilateral cortex causes delay. Such delay is noted in the present study, and Snider's (1950) paper suggests a possible circuitous route to account for it.

A Posterior Parietal Syndrome.

Bender discusses sensory adaptation and after-sensation as peripheral and as central phenomena, and uses the clinical criterion of comparison with the normal side. The patient in his Case I, with left occipito-parietal lesion, showed visual fluctuation and extinction, but the somatæsthetic disturbances were particularly studied. Just as fluctuation and extinction are sensitive tests for central (that is, cerebral) visual impairment, so the sensory adaptation time and after-sensation are demonstrated to be sensitive tests for parietal cortical somatæsthetic impairment. This patient had an embarrassing experience due to imperception for the right hand and forearm. Whenever they were at rest, he was unaware of their position. Also he would suddenly reach for something, almost ripping his pocket off because he was unaware that his hand was in his pocket. Sensory analysis revealed that, while sensibility was intact as judged by the usual routine tests, the sensory adaptation time for kinæsthetic, proprioceptive, tactile, deep pressure and pain stimuli was greatly reduced. When both hands were placed in warm water, the sensation of warmth and wetness rapidly disappeared from the right hand, so that he did not know whether it was immersed or not. Similarly, in tests for stereognosis and barognosis for standard weights on the right hand, he gave accurate answers initially. Then he rapidly lost these faculties, and with the loss came imperception for the right hand and forearm. Similar phenomena occurred with the right leg. Graphæsthesia and all sense modalities were found to be impaired on simultaneous symmetrical stimulation.

Associated with the decrease in sensory adaptation time was a marked decrease or abolition of after-sensation, and it appears that the imperception phenomena were, at least in part, due to these sensory deprivations.

This striking analysis of imperception throws considerable light upon the basis of the body-image and imperception in particular.

An Antèrior Parietal Syndrome.

In contrast to this patient with damage involving the posterior parietal cortex, causing reduced sensory adaptation time and after-sensation, is his second patient, in whom the anterior parietal cortex was involved in a left fronto-parietal cortical lesion, with the production of increased sensory adaptation time and after-sensation. Also of interest is the description of the seizure that occurred three months after injury. It was a right-sided convulsive seizure preceded by a spinning sensation on the right side of the body and face. Another began with twitching in the right arm, followed by paralysis, with which the patient had a sensation of rapid whirling and revolving of the arm, even when it was quite still. In later fits he simply had a sensation of falling to the right. The increased sensory adaptation time and after-sensation were found for touch, temperature, pain and vibration. The stimulus felt more painful on the right side and lasted longer, but often seemed duller on the left. With simultaneous symmetrical stimulation, if the stimulus was stronger on the normal side, then there were reduced sensory adaptation time and extinction on the affected side, with increased after-sensation when the stimuli were removed. If the stimuli were equal on both sides, there was enhancement of sensation on the affected side, with increased sensory adaptation time and after-sensation.

Adaptation.

Bender discusses adaptation, normal and abnormal. He contrasts the apparently uncommon increase in sensory adaptation time and after-sensation with anterior parietal

lesion, with the commoner reduced sensory adaptation time and after-sensation with posterior parietal lesion.

A common normal response in sensory adaptation to pin-prick is the following sequence: sharp intense pain; sharp pain; pain; dull pain; weak pain; pressure; weak pressure; tickle; no sensation. The sensory adaptation time varies from place to place, but is nearly equal for symmetrical points; for example, if it normally takes 80 seconds for a 2·5-gramme weight point stimulus to become unperceived at the right index finger, it may take 70 to 90 seconds at the symmetrical point on the left.

Bender discusses the likelihood that there is an adaptation for the perceptual performances of stereognosis, barognosis and kinæsthesia, with resultant changes in the body-image, apart from the adaptation for the simple sense modalities upon which these perceptual performances are based.

Spatial Visuognosis.

In a comprehensive survey, Bender and Teuber (1947-1948) discuss the spatial organization of visual perception after head injury. They begin with a review of earlier work on such parieto-occipital injuries, pointing out the confusion of the earlier interpretations and the more fruitful work of the *Gestalt* neurologists, especially Goldstein and Gelb. They refer to Goldstein's book (1942) and to the study of spatial visuognosis by Goldstein and Scheerer (1941). Their first patient showed body-image disturbances, particularly a forgetting of the left half of the body unless he looked at it. There was left hemiplegia with sensory disturbances in the somatæsthetic and visual functions. Their second patient had a wedge defect in the left lower homonymous quadrants. These patients were subjected to a battery of tests, of which many were easily applicable to the patient reported upon in this study, without elaborate equipment; they throw interesting light on these sensory functions.

As in the above-mentioned studies, *Gestalt* theory is drawn upon by Bender and Teuber (1948) and by Bender and Kahn (1949) to explain the phenomena observed in the after-imagery in patients with "perimetrically-blind" fields.

A Dynamic Field Theory.

Bender and Teuber (1946) presented a valuable, extensive and detailed paper on fluctuation, extinction and completion in visual perception. In brief, they found that the three phenomena appeared to be related, extinction being the extreme of fluctuation, and completion being the absence of extinction. Whereas prolonged exposure and/or simultaneous symmetrical stimulation produced extinction, brief tachystoscopic exposure produced completion, apparently because the latter condition precluded extinction.

Bender and Teuber point out that since such dynamic processes determine the extent of the functioning visual field, it is absurd to suggest a point-for-point correspondence between function and substrate, as it is commonly held in classical neurology. Different methods of field-taking result in widely different visual field plots.

This variability, characteristic for cortical impairment, was ascribed by Head and Holmes (1911-1912) and by Critchley (1951) to variations in attention. It is explained by Bender and Teuber, in the above-mentioned paper, as due to the lability of threshold in impaired tissues, and in terms of Goldstein's theory that greater energy is required to activate such tissue (*vide* discussion on the paper by Bender and Furlow, 1944, in which Goldstein summarizes his views). There would be a spatial gradient from normal to increasingly impaired tissue, and a temporal gradient related to the duration of stimulus. Therefore, both temporal and spatial variations will alter the functional field.

Bender and Teuber's contention is that fluctuation, extinction and completion are normal phenomena, easily demonstrable only under certain test situations and in certain pathological conditions. They explain many otherwise obscure phenomena, visual and somatæsthetic, including enhancement and extinction on simultaneous symmetrical stimulation, varieties of after-sensation, and monocular enhancement and extinction. They suggest that these three phenomena may also pervade higher functions such as memory and thought.

Extrapersonal Space.

Of particular interest are some clinical discussions in which useful attempts are made to explain a variety of such symptoms as diverse expressions of a common denominator. This is done in *Gestalt* terms according to the vector theory of Koffka and Kohler, and Goldstein's (1939) holistic theory—*vide* the communication by Bender and Wortis (1947). In the discussion that followed this communication, Goldstein, Kluver and Emanual D. Friedmann added to the material, and Friedmann described a case in which the constellation of the left side was so completely obliterated that not only were hemiparesis and anosognosia present, but also the patient was unable to recognize Friedmann when he was standing at the patient's left side. The patient immediately recognized Friedmann as soon as he moved to the patient's right. This was so for both visual and auditory recognition.

In contrast to this complete imperception for the constellation of one side is the exquisitely localized phenomenon of imperception of paresis of the eyelids, and of their position, described by Rubinstein (1941). Of his four patients, the third showed this as the only imperception phenomenon detectable.

Bender and Nathanson (1950), also seeking a common explanation for diverse phenomena, traced uniform patterns for sensory and body-image disturbances. They demonstrated that acral regions are most severely affected; that a widespread disturbance recedes towards the acral regions during recovery; that this disturbance spreads in the reverse uniform way under aggravating circumstances such as thiopentone induction of anæsthesia, and recedes to the acral regions once more as the thiopentone level falls. Their patient displayed pronounced body-image disturbances which extended to extrapersonal space. They referred to Kolb's (1950) report on surgical excision in the parietal lobe.

There are many more papers by Bender and his coworkers that might be cited to indicate the extent and nature of their investigations in the field of temporoparieto-occipital cortical damage and its effects upon somatæsthetic visual and body-image function. These investigations demonstrate the particular importance of the occipital cortex for our orientation in visual space, which is so vital to the integrity of the optic part of the body-image, as described and emphasized by Paul Schilder (1935).

Alexia.

A syndrome of alexia, amnesic aphasia combined with right homonymous hemianopia from a lesion probably involving the left lingual gyrus, described by Klein and Attlee (1948), bears considerable similarity to the syndrome described in the present report. Discussing theories of aphasia, these workers stress that word-blindness occurs in two main forms: as part of an aphasic disturbance of internal language, and on a visual-agnostic basis. "Pure word-blindness" applies to subjects with no defect of internal language or writing. A striking feature in their patient was her ability to indicate the meaning of words she could not read. She said: "I cannot tell what they are, but they come back into my mind afterwards." Also, later, when she could read letter by letter, if she made literal errors they appeared when she produced the word; if her spelling was correct, so was the word. Thus she transferred literal impressions piecemeal into the sphere of language to build a *Gestalt* that she could not achieve by visual gnostic means. Similarly, alexic patients may bridge such a gap by making writing movements with a finger while reading, a manœuvre also used by the feeble-minded and by young children, and helpful in states of severe fatigue.

The foregoing is reminiscent of Schilder's (1935) discussion of Goldstein and Gelb's patients with optic agnosia, in whom, despite the optic agnosia, movements could not be initiated unless the subjects looked at and perceived

the part to be moved, or else made use of preliminary muscle-twitchings (*Tastzuchungen*). Blind people eventually learn to replace these muscle twitchings with kinæsthetic imaginations; the same procedure is used in the "Japanese illusion".

In Klein and Attlee's alexic patient, the word-blindness was mainly of visual agnostic kind; but there were aphasic aspects also. As the authors phrased it, there was dedifferentiation of speech at a certain functional level—terms in harmony with Head's concepts.

Heautoscopy.

Striking examples of the ways in which the body-image can be dislocated into the visual sphere have been described by L'hermitte (1939 and 1951), who gave this phenomenon the term "heautoscopy". Other examples are described by Critchley (1950). This is found most often in schizophrenia and in drug-intoxication, in which the distinctions and boundaries between self and non-self have become weakened and blurred; but L'hermitte (1951) emphasizes that heautoscopy occurs in epilepsy and various organic brain diseases, and should put one on guard to exclude this possibility before regarding it as psychogenic.

Part II of this communication presents the clinical findings in the case of an unfortunate man who suffered a head injury, apparently sustained by a fall from a first-floor balcony. These findings include somatæsthetic, and visual, body-image and speech disturbances.

PART II.

This section presents the clinical findings in the case of a man who was admitted to the Receiving House, Enfield, early in 1951.

The patient, a man in his early forties, was admitted to hospital with a history suggestive of organic confusion, and a vague history of a fall and head injury. A preliminary diagnosis was made of post-traumatic confusion, with right homonymous hemianopia and disturbances of speech and of body-image. He evinced mild vital depression, such as is commonly seen in the tropics, where he had been for some years. This impression was clarified by subsequent investigation, which, because of his expressive difficulties and fatigability, especially early, had to be carried out piecemeal over a period of a month. It was not commenced until he had had time to become thoroughly familiar with his new surroundings.

Thursday Island Hospital Period.

On July 31, 1949, he had sustained a head injury on Thursday Island.

Gnosis.

He recalled a vague memory of being taken to hospital in a motor-car. He forgot everything that had happened from 6 p.m. on July 31 until about midnight, when he was in this car.

"I seem to have woken up while the car was travelling to hospital. I woke up just enough to see that I was in a car and to know it was travelling. I seemed to say to myself 'I'm being carted off to hospital'. I seemed to think I'd broken myself, or my bones, or something. I didn't see anything that I remember; but it was dark, in the middle of the night. I must have slept again until I got to the hospital. I saw and recognized the doctor, whom I'd seen recently, and I remember his talking to me and asking questions. The only thing I remember of what he said was 'Now you must go to bed', and repeatedly asking 'Do you understand that?'. I thought I was speaking clearly, but apparently I wasn't, because he kept asking if I understood until I nodded. I could understand what was being said, but apparently was not speaking clearly. I recognized that I was in the hospital, but I didn't know what part of the hospital, and, although I know I wasn't standing, I don't know whether I was lying on the floor, or partly lifted, or what. I just remember seeing the doctor and realizing I was in hospital. I must have fallen asleep again then."

"The next thing I remember was saying to myself 'I'm ruined now, how to live. There's no job I could ever do again', half thinking and half dreaming. I don't know whether it was then light or dark or whether my eyes were open or shut. This seemed to go on for an hour or two. I was in bed."

"I imagine it was about a day after that a man woke me up by shaving my head. From this time on, although I slept a lot in the ordinary way, I was wide awake and aware of what was going on around me. It was now daytime, but I don't know what time of day. I think it was the next day that the doctor and matron came around at about mid-morning. I spoke clearly in reply to what the doctor asked, although I can't remember now what he asked; I understood at the time and have forgotten since. When I replied to the doctor the matron said 'The man is a bit mad' or 'The poor bloke's mad', or something like that. The doctor said 'No' as they walked away and he seemed to be explaining to her as they walked away and out of the ward. I could see everything about me, but it was unfamiliar, as I'd never been in the ward before, although I'd done some jobs in other parts of the hospital.

Perhaps about three weeks later, when I was allowed up in a self-propelling wheel-chair and went out of the ward, whenever I came to part of the hospital that should have been familiar, I thought to myself 'That's strange, and not quite as it ought to have been'. I thought it might have been the other hospital—one that was never used, as it was the quarantine hospital, and I'd never been in that. It was the same with everything else where I had to use my eyes: I didn't seem to be seeing normally. If it hadn't been for this I should have known where I was in the hospital. For example, I picked up a jam-tin and although I could see it clearly and could see printing on the label, and although I knew they were letters of the alphabet, I could not recognize them or read the words, no matter how big or clear they were. For example, there's one brand of jam, "I.X.L.", on which those letters "I.X.L." are very big and clear. Seeing that there were three big letters on the label I could then know that it was that one brand of jam, although I could not think or say 'That's I.X.L. jam'."

At about this same time (three weeks after injury) he also noticed that he could not see so far around to the right as to the left. Then he noticed that, when doing jobs in the garden, for example, he would tend to turn to the left. If he was weeding and set out to weed in a straight line across a bed, he would veer to the left instead of advancing in a straight line.

Subjective Sensory Phenomena.

Since Awakening in Hospital.—"There's slightly funny feeling, in any case, when it doesn't move, and I couldn't move at all." "It felt as though something was dead, some sinew I should imagine, all the way up the outer side of the right foot, leg and thigh, and here on the right arm"—demonstrating the ring and little fingers and the ulnar aspect of the hand, wrist and forearm, up to the elbow. "It is a loss of feelings." "That is how it was then, and it still is like that. It's not natural. It's natural to feel everything." Apart from this numbness there was no anæsthesia or paræsthesia at the time. "Any tingling?" "No, not then."

Five Months After the Accident.[1]—"That word 'tingling'. I've been trying to think of the word to express that feeling. I know I'd said something to the doctor in Darwin. He asked if it was an ache. I said 'No, it's not an ache', but I could not think of the right word. I have had this tingling since five months after the accident, and I get it nearly every time I put my head down for half an hour or so or for the night. I've just had a sleep sitting on the sofa, with my head on the back of the sofa. When I woke up it was bad." ("How bad?") "It's hard to say. When that happens I'm saying to myself 'I'm ruined if that can't be squared up'." ("Why ruined?") "Well, the feeling is something—I think I'm going to faint or die, or something." "Whenever I wake up, whether I've had enough sleep or not, I have the same feeling as I used to have if I needed more sleep. Of course I do sleep too much. I sleep any time during day or night. I'm not correctly awake after sleeping; it is like being in a dream, and there is a sort of ache in the head. It's always a bit like a dream. I would need some time to realize, to calculate 'this is important, or not important', instead of seeing at a glance. If I was sweeping and came to that doorstop there, I'd have to spend four times as long to consider it, instead of sweeping past it. It's always much worse on first waking, and this feeling that it's got to be squared up. Things about mental is hard for me to know about, but I don't like the idea that mental disease is mine. I think I really feel the brain inside the head, moving. I didn't know about the body, before, that I know now. I can feel the pulse in the head. The head feels as though it's got a tight hat on, now and again. This moving feeling is when I lie down, and again when I get up, but it soon gets right again—after a little while. It's like a lift feeling—

[1] Quoted *verbatim* carefully to accentuate the aphasic speech.

when the lift stops quickly, something goes up out of the head, and then comes back again. But mine takes a little while to come back, slowly. When I wake up, I have to square up everything about me: the mind must square up and feel every part of the body. People get excited and sit down beside a chair and miss it; it takes me a little time to see where I am going to sit and just how and where to move that part of the body, or I will miss the chair too. I feel that the parts of the body are disconnected: they are lying apart without any life, by me. When I wake up, I have to think about it, then I have to feel out in the parts and square them up. Then, after some time, I begin to feel they are all me, and I feel fit. Moving helps, but I have to move slowly, because it becomes bad when I move rapidly, and I have to hang on to something that doesn't move. This happened on a moving truck, too. It's all a bit like it was when I used to wake, after not nearly enough sleep, before the accident, but it's more sharp now, and lasts quite a bit." "It seems very serious." ("Is it giddiness?") "Giddy is the word I use for all the time of every day. I think it's only because I'm used to it that I can walk and move. I see better—I see twice as well when I sit without any movement, as when I am moving about. Whenever I move I don't see as well. For example, I noticed particularly this morning, after walking say from here to there, when someone brought up a cup of tea I was looking only at that cup of tea. Then I saw a fellow, and thought, 'Oh him!' I hadn't noticed him, although he gave me the cup of tea. The same with others who were in the group of chaps there. I'd see only the face, not the rest of him. You know ,if there's something serious, you look just at that, but if it's not serious, you see other things around. I don't see the other things around." ("Tell me more about this giddiness.") "I'll have to think it out what it is when I say 'I'm ruined'. I'll have to think it out what it is. For example, there's a sort of taste, a sudden taste in the tongue and the lips: an electric taste. Not an electric shock, but the taste you get when you put your tongue on the brass top of a torch battery." ("Metallic?") "Yes." ("Why didn't you say so?") "I was just thinking of a part of a battery—volts. I wouldn't have got that word until you spoke it. I've had to wait for people to say words before I could use them again." ("Where do you get this taste in the lips and mouth?") "In the front. In the — I just can't get the word for it. In the front of the tongue. I think that's exactly what I mean—the front of the tongue." ("The tip?") "The tip? Yes. That's the word. I hadn't thought of it." ("That describes it better than 'the front', does it?") "Yes. That's just the word. I've worked it out now, it's not the lips, only the tongue."

There had been no change in the subjective sensory phenomena since the time when he first recognized them—the numbness a few days after the accident, the visual disturbances about three weeks after it, and the paræsthesiæ and giddiness five months after it.

The "giddiness" was not vertigo; the room did not revolve, nor did he. There was no rolling or pitching. But when he turned his gaze towards a small object, that object seemed to travel part-way to meet his gaze, vertically or horizontally, or obliquely, in exactly the opposite direction in the same line of movement.

("Are you getting wearied?") "It's hard for me to listen to what you say." ("What's the difficulty?") "Apparently whatever I think for myself I can hear properly. I don't easily hear what some other person speaks of, something that I'm not concerning myself with; but I'm not getting weary. I could go on."

Motor Phenomena.

For the first few days in Thursday Island Hospital he could not move at all. "I realized that immediately, when I was in the car. I should imagine I was broken in every bone. If I'd had the habit of child I would have cried, because I was saying to myself 'I'm ruined' and imagined that everything horrible had happened to me. It didn't stop until I'd worked out what I could do. I was happy then, for some while, when I worked it out I could be a lighthouseman. I did this when I could move again. And yet I must have moved, because I used to fall out of bed. I wouldn't move for about two weeks, even after I could speak quite well. I then got out of bed." ("Tell me about your first attempts to move.") "I tried straight away, but found I couldn't move. They used to put food into my mouth—oh, one side moved perfectly—the left side—after the first few days. The right hand and leg began to move after about two weeks. I first moved the whole limb, then had to learn to move the fingers and toes. I didn't think of it, learning. I would decide to make a movement and then do it; but I would forget about it. It worried me about why this arm didn't work properly. I realized that when I forgot about it, it wouldn't move. I got into the habit of using this one [left arm] instead of this [right arm]. I would actually forget that the right arm and right leg were there. It is the very first thing I remembered after waking up properly, that the right arm and the right leg feel in some way dead, just as normally an arm feels dead after you've been sleeping on it for a while. I forgot to use them, and I had to get into the habit of using the right side again. I imagine that I thought it would grow natural again; but after it had got so well, and no further, I realized I had to make it better myself, and so I began practising using the right side. I could still easily forget that the right side could move, while I was practising. Even now I still have the idea some sinews are missing or broken.

"About three or four weeks after accident, I got as far as doing up buttons. I would find myself doing it with my left hand, and think 'Oh no, I must do it with my right hand'. It was clumsy for a long time; more than two months. Then and since, if I was holding something and talking to someone, I would easily forget, and drop what I was holding with my right hand, not the left hand. It was more the difficulty of *remembering* to use my right hand than actually *using* it. I had to teach myself again, every movement of the fingers, for example, in doing up a button; but, even then, it was more difficult to *remember* to use my right hand than to *use* it."

"After two weeks I was able to get into a chair, and within another week I could walk. It seemed that almost as soon as I could move a limb, I could use it. It was quicker than anybody thought I would be able to. The doctor was surprised."

"I must have spoken within a few days, but until then I did not say one word that they knew; but I could say any word that came to my mind, and as soon as I heard words I could repeat. I didn't slur. They could hear and understand me."

Subsequent History.

He left Thursday Island five and a half months after the accident—that is, in January, 1950—and went to Darwin for most of the year, until mid-November.

"Most of the time I went to the municipal job for the sweeping. They saw I was speaking intelligently and gave me better jobs, building up little shacks and everything. We done some water-pipes."

He was in Darwin Hospital on three occasions; he woke up in the hospital after some faints. On the third occasion he was in hospital for a month. "Nothing seemed to make any real difference"—meaning that there was nothing different in his state. "I thought perhaps my mind was ruined. I couldn't write or read well, because I couldn't think of what I was writing or reading." "I then went out and sat in the bush, because I thought I could easily lay down and die. There was not good point for living. As a matter of fact, it was the mosquitoes that prevented that." He was offered a lift in a passing truck. "He was going to bring me right to Adelaide; but I had to wait a few days for him, so I jumped on another truck and got to Alice Springs." The doctor advised him to stay in the Alice Springs hospital a while. He stayed seven weeks, waiting while the doctor obtained more information from Darwin. Eventually he was certified insane.

The following conversation between an escort and the patient prior to leaving Alice Springs throws further light on the history and the nature of his mental defects.

"What is your name?" Correct reply.

"How long have you been here?" "About a week."

"Where were you before?" "I walked out of the Darwin hospital."

"Why do that?" "I wanted to destroy myself but the mosquitoes wouldn't let me."

"What do you mean?" "I thought I could lie down in peace, but the mosquitoes would not let me."

The escort added: "During the time I was questioning him he was looking at me quietly and blankly. His answers were vague and it took him some time before he could answer a question."

Two doctors examined him. One reported as follows: "He is abnormally slow in answering questions and appears to be confused by simple questions. His memory is deficient in nearly all details of small or important events in the last two years." This doctor added that the patient had been walking aimlessly about the hospital talking to himself and was incapable of performing any but simple functions. The other doctor added that he was unable to care for himself and was unable to remember words in a conversation. He

was slow in his movements and speech and answered questions irrelevantly. He stood unmoving for considerable periods of time, and when asked why he was doing so, was confused and unable to explain; he was confused as to his location and as to his purpose in being in the hospital grounds.

On arrival in Adelaide, a further conversation between escort and patient was as follows:

"What is your trouble?" "I ruined my brain a year ago. I broke the head."

"How?" "A fall."

"What was the result of the fall?" "Apparently nothing else, only a tiny cut in the head. It stopped half of me, half of the body, half of the eyes."

"Does it cause you any trouble?" "I have trouble getting about and making my way in the world."

"How long have you been like this?" "It's soon as I got out of the hospital. I didn't know what was wrong. Perhaps something isn't quite right in the brain. I am all right when I sit down, but when I stand up something goes wrong and I go giddy. Sometimes I don't know what I am doing."

"What trouble do you find mostly in life?" "I don't know how I am going to live. There are many things I don't see."

On his arrival at the Enfield Receiving House, early in 1951, the patient was moderately depressed and retarded, somewhat apathetic. He was mildly confused, becoming fatigued, perplexed and muddled on being questioned. Right homonymous hemianopsia was apparent; but, after preliminary examination it was decided to defer close investigation until his general state improved, then to proceed piecemeal. The depression, retardation and apathy seemed to be typical of that state of mind, common in the tropics, which is usually referred to in such places as "troppo", and which was fairly common in servicemen who were inadequately occupied in garrison areas (for example, Darwin) during World War II. This has decreased considerably since his admission to the receiving house, but he is still hypokinetic.

Neurological Findings.

Preliminary Findings.

Examination of the right optic disk revealed that it was a little reddish but otherwise normal, with cribriform pattern and margins clearly defined. The left disk was a little pale and bright, the trunks of the main vessels being reduplicated. The cribriform pattern was not so clear and there was some greyish blurring of the naso-superior margin of the disk. The veins were rather full and pulsation was more prominent. Otherwise no abnormality was detected. Examination of the fields disclosed right homonymous hemianopsia.

Muscle development, tone and power were good. The reflexes, superficial and deep, were equal and active, except that the left adductor reflex was weak, and Hoffmann's sign was present on both sides. No *tache cérébrale* was present.

No abnormality was detected in cerebellar function.

Investigation of sensation revealed some subjective abnormality over the lateral aspect of the right foot and thigh.

Cranial Nerves.

The function of the cranial nerves was investigated, with the following results.

First Cranial Nerve.—He could taste and smell food, drink, tobacco, flowers *et cetera*, but frequently he had a spontaneous smell and taste of dirty diesel oil, which was there when he awoke, usually lasted two days, and was gone on the third day. He noticed it most when smoking, so that the first smoke of the day was a critical test for its presence; but it was also there when he was eating or drinking, and he could notice it at any time on those days. He smelled and tasted the normal aromas and flavours, but the smell of oil was there too, and was especially strong when he was smoking. It reminded him of his experiences just before his accident, when he complained of the foul aroma and taste of the tobacco aboard a pearling lugger, until it was explained to him that the diesel oil fumes, which pervaded the lugger, affected everyone that way.

Second Cranial Nerve.—He could see clearly (near and distant vision) and could see small print clearly; but he had difficulties in perception and understanding (*vide infra*). Investigation of the visual fields revealed right-sided homonymous hemianopsia, stopping accurately in the vertical plane of the optic axis. He had noticed this first soon after the accident, when he could see objects further out in the periphery of the left field of vision than in the right. When he was tested, he realized that the whole right field was missing. Confronting one and fixing his vision on the top of one's nose, he could not see one's left eye. He was worried by flashes of light when turning from bright to dark.

Third Cranial Nerve.—The left pupil occupied half the iris and the right pupil one-third of the iris. The pupils were round and active to light, directly and consensually, and to accommodation and convergence. Good mydriasis was obtained.

Third, Fourth and Sixth Cranial Nerves.—On convergence, double vision appeared at four inches from the nasion; but on being tested with the ophthalmoscope light the reflex moved from the centre of the right pupil at five inches to four inches, wandering nasad as the right eye drifted temporad.

Fifth Cranial Nerve.—Motor and sensory functions were intact.

Seventh Cranial Nerve.—Voluntary and mimetic functions and the reflex were intact.

Eighth, Ninth, Tenth, Eleventh and Twelfth Cranial Nerves.—All functions were normal.

Sensory Function.

Touch and vibration sense was present, but of variable sensitivity. There appeared to be impairment of appreciation of deep pressure, pain, heat and cold on the whole of the right lower limb, from the ilio-sacral region down, and of stereognosis and two-point discrimination, but not of point localization, in the same limb. On closer examination it was found that the quality of the sensation was different, rather than the intensity, between the two lower limbs. Elsewhere it was normal and equal.

In the lower limbs deep pressure was felt as touch on the right, as pressure on the left; the pressing object could be identified on the left, but not on the right. Pin-prick was recognized as sharp, without any feeling-tone or emotional response on the right; normal painful sensation and response occurred on the left, on which side pin-prick was felt to be unpleasant. Warmth was recognized on the right as warm, without any feeling tone or emotional response; normal warm sensation with pleasurable feeling-tone was present on the left. Exactly parallel responses to cold stimulus were present; on the left they were normal, on the right abnormal.

The left leg felt cold, subjectively (cold day), while the patient knew that the right leg was cold only when he felt it with his hand, and he had the cold sensations in the skin of his hand, not in the leg. "The right leg feels dead and lifeless, and as though it doesn't properly belong to me. It doesn't feel as though it's got weight. It doesn't get uncomfortable in bed, so I don't have to move it about, unless it presses on the left leg. It doesn't make worse if the left leg presses on the right." I pressed on his right leg and he said he felt my hand touching his leg, but he did not feel pressure or discomfort, although I was pressing hard on his knee, hyperextending it as well as pressing it firmly into a firm couch. Stereognosis was not very good with either foot and toes; but it appeared to be more a general cognitive disturbance than a sensory impairment, because he eventually described the hardness and thickness correctly, even though he could not identify the object (compare bilateral astereognosis due to left supramarginal lesion). The right lower limb, and to a less extent the right arm, felt and behaved differently. There was a feeling of lifelessness, and of their not properly belonging to him. In the lower limb he felt "like a dead tendon, like a string, down here and here", indicating a line running down the antero-lateral aspect of the calf and foot. In walking he felt the left leg moving, with kinæsthetic and pressure sensations, but he knew only that the right leg was walking, more like a wooden limb. The right arm did not swing unless he reminded himself of it and swung it.

When he was sitting he unconsciously made small muscle and joint movements, as normal, on the left side; but the right arm and leg stayed almost inert. He said that in the cold weather his left arm and leg soon warmed up when he was walking or even standing or sitting, but the right leg particularly and the right arm stayed cold. He had noticed that in the preceding few days, the first cold weather he had experienced since his accident. This had brought to his notice that the right arm and leg were not automatically making those small movements, whereas his left limbs were. Once he had thought of it, he could make those small movements, following with each individual movement after thinking it, then trying it. Previously it had not occurred to him to make them. (Early after the accident this was true for even the simplest large-joint movements.) Even so, there was not the automatic play of small movements that was present on the left.

He always crossed the left leg over the right, because he did not feel any uncomfortable pressure on the right leg, as he did on the left leg if he crossed the right leg over it. When sitting with legs parallel and both feet resting on the floor, he felt only contact-touch in the right sole, but both touch and pressure in the left sole. Moderate weight on the right knee was still felt only as contact-touch at knee and sole; but heavy weight on the right knee caused some sense of pressure in the sole. When he crossed the left leg over the right he forgot about the right leg; it felt as though the left leg was crossed over some other object. Similarly, when he was lying in bed or when he was resting quietly he forgot about the right leg. He simply put it in some position: "I put that thing there like that. It just stays there. I have to make the left leg comfortable, though, and move it about occasionally, to keep it comfortable. The right leg is probably where I put it when I went to sleep; when I wake up it hasn't moved. It is lifeless. I feel and know my left leg, and it moves itself to keep warm and comfortable; but I have to think it before I can make the right leg move." "I am afraid that I can only imagine that small tiny muscles or sinews never move, unless I know and think to move. Similarly, I had to *learn* to move of the more simple bigger important muscles. Even now I do not move the right arm, as I walk, unless I suddenly notice that the left arm is swinging naturally."

Present Status of Speech.

Visual Considerations.—Right homonymous hemianopsia creates mechanical difficulties. He can read a single line easily, but when he is confronted with a page of print he finds that, because he can see only to the left of the mid-line, he can see only the first word of each line. Consequently he has some difficulty in finding the correct line after the first few and until the last few on the page. For this reason he finds it rather easier to turn the book clockwise through 90° and read the line vertically from top to bottom. Visual dysgnosia is present (*vide supra*—for example, his difficulties with the "I.X.L." jam label and his limited awareness in the peripheral fields). Apart from the fatigue occasioned by these difficulties he can read and understand quite normally and has a good memory and general grasp of what he has read. He can read a map and measure a distance and direction between two points upon it.

Auditory Considerations.—His hearing is good and he understands everything said to him, provided that it is not too rapidly spoken, and provided that it is spoken reasonably clearly and in good English. Auditory dysgnosia is present (*vide supra*—for example, his impaired grasp of topics not currently in his mind).

Writing.—He can write and print easily, fluently and legibly, spontaneously, on dictation or by transcription, so long as he does not have to retain more than one or two words in his mind at a time before writing them. If he is given a whole sentence he can grasp the sense immediately, but he cannot retain the words; for example, "the provision of running water is an essential" was paraphrased "it is very necessary in any case that there must be sufficient water". He can write simple arithmetical propositions; but he could not recall how to write "Y^2" nor could he answer the question "If $Y^2 = 16$, what does Y equal?" He said: "I used to do this though."

Speaking.—He shows the same disturbance in immediate retention when asked to repeat a sentence that he had read or heard immediately before. He finds it hard to recall names of people, except those of special importance and familiarity, such as the doctors' names. After his accident he lost all his speech and slowly acquired it again from hearing the speech of others. He still finds that he cannot produce a word that he knows exists—for example, the names of the compass points—and it was only when he read it in the paper that he could say the name of his birth-place, although he could describe the town and all about it. He did not notice any difficulty in syntax or in semantics. "As soon as the words came I was able to use them freely."

When it was pointed out that he had said "I was ruined how to live?" he recognized that it was clumsy syntax and then substituted "It is impossible, the correct life". He again recognized the shortcomings of the sentence and said: "This unhappy life I can no longer live correctly." He welcomed my suggestion, "I can no longer live life to the full", as expressing the sentiment precisely. He has no loss of non-verbal language, spontaneously or on\ request, although he is generally constrained and reserved. His chief difficulties in speech, then, are neither sensory nor motor, but amnesic, and, while his aphasia or speech difficulty is most prominently verbal and nominal, it is also syntactic and semantic. His difficulties are increased by some slight impairment of grasp and comprehension, and by moderate impairment of immediate retention, although his memory in general is good, apart from the concussion amnesia. He speaks slowly, with effort, always seeking, and seldom quite finding, the normal way of expressing himself clearly.

General Gnosis and Praxis.

There is some autotopagnosia for the right limbs all the time and some acalculia; but there are no other components of Gerstmann's syndrome. He has general autotopagnosia on awakening.

He has no anosognosia; there are strange sensations and an alienation of the right lower limbs, and to a less degree of the right upper limbs, but there is no failure to recognize disabilities.

He has no apraxia, object or reflectory, no finger apraxia, constructional apraxia, eating apraxia or dressing apraxia, either actual or in mime, whether on request, on imitation, spontaneous or on semi-automatic motivation.

There is some giddiness, which appears to be related partly to his visual dysgnosia, partly to a sense of dispersal of the body image, and partly to the "lift feeling", the emanation from the head.

Some tingling paræsthesia of the tongue-tip and of the limbs on the right side is present, with hypæsthesia and delayed sensation but no alloæsthesia.

This picture has shown minor variations, in many of its parts, from time to time; but mainly the difficulty has been for him to express himself clearly about experiences that are strange to him and intangible.

Findings at Subsequent Examination.

In May, 1951, examination revealed that the optic fundi were *in statu quo*; no abnormality was detected. With eyes closed he could imagine and point to familiar landmarks.

He still tended to veer to the left when digging or weeding across a garden, walking where there was no well-defined line (for example, a kerb) to aid him. He had to attend constantly to avoid this veering, though it was only slight and not apparent—for example, in walking across a room, over short distances.

Goldstein's match-stick test was done easily and rapidly. He recognized tri-dimensional drawings on a sheet of paper and obtained the usual reversal phenomena as with the Schroder staircase illusion. He copied, then drew from memory, a tent, an open book and a cube.

He could recognize and read a map, finding the distances and direction from one town to another, using the scale. He drew a rough sketch, to scale, to show how he would go from the office along three passages and across an open space to the wood-heap, with directions and distances and chief landmarks quite well shown.

Visual imagery, after-imagery and memory were good for form and colour.

Light perception in the perimetrically blind field was tested in total darkness according to the method described by Bender and Krieger (1951). After some hours in total darkness he was given a fixation point directly ahead by an attendant's clicking his fingers. A standard "Keeler Senior Model Ophthalmoscope", suitably guarded, was used to provide the target light, exposed at 60° of arc from the visual axis, first in the right hemianopic field, then in the left normal field. There is a round illuminated window, four millimetres in diameter, at which the dioptre reading shows. This will be called "A". Below it is a round illuminated hole 1·5 millimetres in diameter; this will be called "B". The reflected beam of the ophthalmoscope was used, with the green filter, giving a disk of green three millimetres in diameter on the ophthalmoscope mirror. This will be called "C".

On the right side, at 60° from the visual axis, and in the horizontal meridian, "A" was seen as a faint, whitish, diffuse glow, as of a fog illuminated faintly by an unseen source of light—like the first faint light of dawn in the sky. He could not point to the target, but the glow ceased when his finger passed in front of the light. "B" was seen as a soft, very blurred patch of light, of no definite size or shape, and unmoving, to which he could point fairly accurately—like a headlight indistinctly seen through heavy fog, whitish or yellowish. "C" was seen as "A" was. There was no difference in colour, and intensity was about the same.

On the left side, at the symmetrically opposite point, "A" was seen as the larger disk, "B" was seen as a pin-point, and "C" was seen as the same larger disk, but greenish, and the colour became more distinctly green as it was brought nearer the visual axis. He could point accurately to "A", "B" and "C". At no time on the left did he see the faint, diffuse glow seen with "A" and "C" on the right.

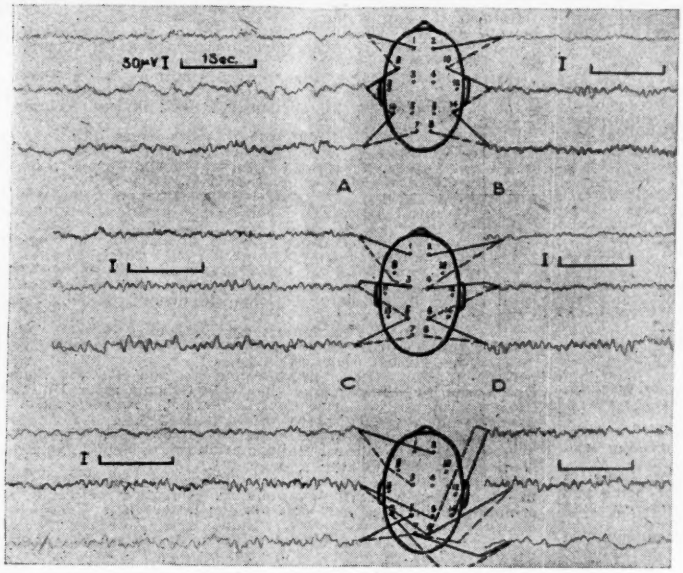

FIGURE I.

He then volunteered that he now knew the explanation of something that had puzzled him previously. He had noticed that, lying in bed at night, he had been aware of a street light, in the middle distance, on his blind side. His room had to be quite dark for this; if the passage light was on, he was not aware of this street light unless he brought it to his left field.

Electroencephalographic Findings.

Electroencephalography early in May, 1951, confirmed the clinical diagnosis of left parieto-occipital lesion, deep enough to involve the left optic radiation, probably deep to the angular gyrus, sustained months previously.

Specimens from the tracings are shown in Figures I and II. The seventh placement is five centimetres above the external occipital protuberance and 2·5 centimetres from the mid-line. The first placement is at the hair margin, four centimetres from the mid-line. All other placements are in straight lines separated by equal distances.

It will be seen that the focus from which abnormal rhythms were obtained is over a wide area. With the bipolar recordings shown there is asymmetry of the voltages from corresponding positions on the two sides of the head, those from the left side being generally larger and slower than those on the right. Low-voltage Δ discharges are present in the lateral leads from the left parietal and occipital regions, but are not present in leads from near the mid-line. There is no phase reversal in this δ activity in simultaneous records from the parieto-occipital region. The α activity in the posterior occipital leads appears normal with higher voltages on the left than on the right. Thus the abnormal activity in the left parieto-occipital region appears to arise from a deep-seated focus.

Results of Other Investigations.

On May 12, 1951, the blood failed to react to the Wassermann test. Lumbar puncture produced clear and colourless cerebro-spinal fluid under a pressure of 135 millimetres of water. The Queckenstedt test produced a normal response. The cerebro-spinal fluid contained one lymphocyte per millilitre, the protein content was 40 milligrammes *per centum*, and the fluid failed to react to the Wassermann test. No precipitation occurred in the Lange test.

As has been shown by Bender (1946), there is a characteristic pattern of response to simultaneous symmetrical stimulation and to tests for after-sensation and sensory adaptation time, in cases of posterior parietal lesion. Such a characteristic pattern was found in this man.

Neurosurgical investigations were carried out by Dr. Leonard Lindon at the Royal Adelaide Hospital. A ventriculogram on July 4, 1951, showed that the posterior portions of the bodies of the lateral ventricles were filled. The right side appeared larger than the left and there was some displacement to the left. There was no filling of third ventricles (the significance of this was uncertain). On August 2, 1951, the insertion of a needle into the left occipital lobe showed that the posterior pole of the ventricle was displaced laterally and that medial to this was some tough fibrous tissue.

Comment.

Detailed investigation of such patients is time-consuming and requires painstaking persistence not always possible in general practice. The importance of referring them to centres where they can be searchingly examined lies not only in the advantages to the patient that accrue from refinement in diagnosis, prognosis and treatment. Each clinical problem of this kind may contribute something to the elucidation of an obscure field.

Even in the last four months of the two-year period following the trauma, slow but progressive improvement was noted; but this was in his spontaneity, responsiveness, and increasing verbal and ideomotor repertoire. The objective neurological findings were unchanged.

This improvement can be largely ascribed to his new cheerfulness and confidence, which came from the sympathetic encouragement and understanding of the nursing staff, and from his growing knowledge of the specific nature of his limitations and of ways of overcoming them. His previous bleak despair is apparent from his own story.

Acknowledgements.

To Dr. H. M. Birch, Superintendent of Mental Institutions of South Australia, I am indebted for the time and care taken in making the electroencephalographic studies and for permission to publish this communication. Dr. J. H. Barnes, of Thursday Island, by his kindness in furnishing the report of his findings in the early post-traumatic

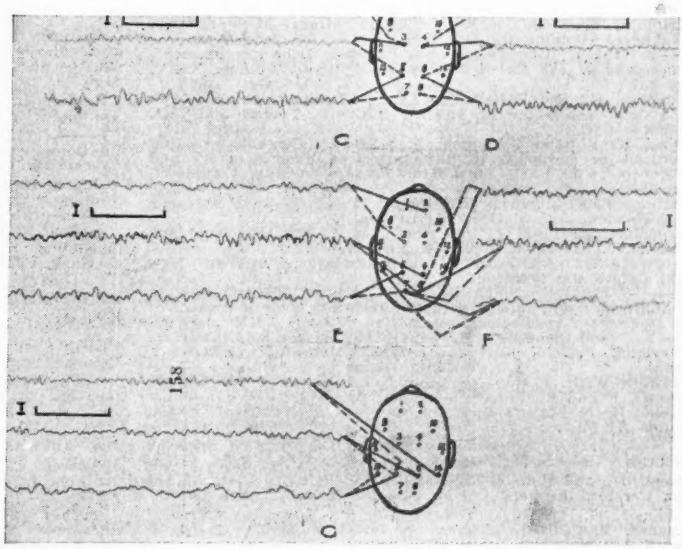

FIGURE II.

period, has filled in a hiatus in the history and lent confirmation to the subsequent conclusions.

References.

Ades, H. W., and Raab, D. H. (1949), "Effect of Preoccipital and Temporal Decortication on a Learned Visual Discrimination in Monkeys (Macaca Mulatta)", *Journal of Neurophysiology*, Volume XII, page 101.

Bailey, P. (1933), "Intracranial Tumours", Baillière, Tindall and Cox, London, page 296.

———, *et alii* (1943), "Functional Organisation of Temporal Lobe of Monkey (Macaca Mulatta) and Chimpanzee (Pan Satyrus)", *Journal of Neurophysiology*, Volume VI, page 121.

Bender, M. B. (1946), "Changes in Sensory Adaption Time and After Sensation with Lesions of Parietal Lobe", *Archives of Neurology and Psychiatry*, Volume LV, page 299.

Bender, M. B., and Furlow, L. T. (1944), "Phenomenon of Visual Extinction in Homonymous Fields and Psychologic Principles Involved", *Transactions of the American Neurological Association*, Volume LXX, page 87.

——— (1945), "Visual Disturbances Produced by Bilateral Lesions of Occipital Lobes with Central Scotomas", *Archives of Neurology and Psychiatry*, Volume LIII, page 29.

Bender, M. B., and Kahn, R. L. (1949), "After-imagery in Defective Fields of Vision", *Journal of Neurology, Neurosurgery, and Psychiatry*, Volume XII, page 196.

Bender, M. B., and Krieger, H. P. (1951), "Visual Function in Perimetrically Blind Fields", *Archives of Neurology and Psychiatry*, Volume LXV, page 72.

Bender, M. B., and Nathanson, M. (1950), "Patterns in Allesthesia and their Relation to Disorder of Body Scheme and Other Sensory Phenomena", *Archives of Neurology and Psychiatry*, Volume LXIV, page 501.

Bender, M. B., and Teuber, H. L. (1946), "Phenomena of Fluctuation, Extinction and Completion in Visual Perception", *Archives of Neurology and Psychiatry*, Volume LV, page 627.

——— (1947), "Spatial Organization of Visual Perception following Injury to Brain", *ibidem*, Volume LVIII, page 721.

——— (1948), "Spatial Organization of Visual Perception following Injury to Brain", *ibidem*, Volume LIX, page 39.

Bender, M. B., and Wortis, S. B. (1947), "Patterns in Perceptual, Motor and Intellectual Functions in Organic Brain Disease", *Transactions of the American Neurological Association*, Volume LXXII, page 31.

Blum, J. S., Chow, K. L., and Pribram, K. H. (1950), "A Behavioural Analysis of the Organization of the Parieto-temporo-preoccipital Cortex", *Journal of Comparative Neurology*, Volume XCIII, page 53.

Bonin, G. von (1941), "On Encephalometry; Preliminary Study of Brain of Man, Chimpanzee, and Macaque", *Journal of Comparative Neurology*, Volume LXXV, page 287.

Bonin, G. von, Garol, H. W., and McCulloch, W. S. (1942), "Functional Organization of Occipital Lobes", *Biological Symposia*, Volume VII, page 165.

Brain, W. R. (1941), "Visual Disorientation with Special Reference to Lesions of Right Cerebral Hemisphere", *Brain*, Volume LXIV, page 244.

Chow, K. L. (1950*a*), "Effect of Partial Extirpation of Posterior Association Cortex on Visually Mediated Behaviour in Monkeys", Comparative Psychology Monograph.

——— (1950*b*), "A Retrograde Cell Regeneration Study of the Cortical Projection Field of the Pulvinar in the Monkey", *Journal of Comparative Neurology*, Volume XCIII, page 313.

Clark, W. E. Le Gros (1948), "Connexions of Frontal Lobes of Brain", *The Lancet*, Volume I, page 353.

Critchley, MacDonald (1950), "The Body-Image in Neurology", *The Lancet*, Volume I, page 335.

———(1951), Discussion on "Parietal Lobe Syndromes", *Proceedings of the Royal Society of Medicine*, Volume XLIV, page 337.

Cushing, H., and Bailey, P. (1928), "Tumours Arising from the Blood Vessels of the Brain", Baillière, Tindall and Cox, London.

Dow, R. S., and Anderson, R. (1942), "Cerebellar Action Potentials in Response to Stimulation of Proprioceptors and Exteroceptors in Rat", *Journal of Neurophysiology*, Volume V, page 363.

Fulton, J. F. (1949), "Physiology of the Nervous System", Third Edition, Oxford University Press.

Goldstein, K. (1939), "The Organism. A Holistic Approach to Biology", American Book Company, New York.

——— (1942), "After Effects of Brain Injuries in War", New York, Grune and Stratton.

——— (1944), "Significance of Mental Disturbances in Rehabilitation of Soldiers with Brain Injury", *Transactions of the American Neurological Association*, Volume LXX, page 22.

———, and Scheerer, M. (1941), "Abstract and Concrete Behaviour", *Psychological Monographs*, Volume LIII, Number 2, page 1.

Gowers, W. R. (1893), "A Manual of Diseases of the Nervous System", J. and A. Churchill, London, page 739.

Head, H. (1923), "Speech and Cerebral Localization", *Brain*, Volume XLVI, page 355.

——— (1926), "Aphasia and Kindred Disorders of Speech", Cambridge University Press.

Head, H., and Holmes, G. (1911-1912), "Sensory Disturbances from Cerebral Lesions", *Brain*, Volume XXXIV, page 102.

Holmes, G. (1918*a*), "Disturbances of Vision by Cerebral Lesions", *The British Journal of Ophthalmology*, Volume II, page 353.

——— (1918*b*), "Disturbances of Visual Orientation", *ibidem*, Volume IV, page 383.

Holmes, G., and Horrax, G (1919), "Disturbances of Spatial Orientation and Visual Attention, with Loss of Stereoscopic Vision", *Archives of Neurology and Psychiatry*, Volume I, page 385.

Horrax, G. (1923), "Visual Hallucinations as a Cerebral Localizing Phenomenon; with Especial Reference to their Occurrence in Tumours of the Temporal Lobes", *ibidem*, Volume X, page 532.

Klein, R., and Attlee, J. H. (1948), "Syndrome of Alexia and Amnesic Aphasia; Subarachnoid Hæmorrhage, with Symptom of Partial Occlusion of Spinal Subarachnoidal Space", *British Journal of Mental Science*, Volume XCIV, page 59.

Kluver, H. (1936), "An Analysis of the Effects of the Removal of the Occipital Lobes in Monkeys", *Journal of Psychology*, Volume II, page 49.

——— (1937), "Certain Effects of Lesions of the Occipital Lobes in Monkeys", *ibidem*, Volume IV, page 383.

Kluver, H., and Bucy, P. C. (1938), "An Analysis of Certain Effects of Bilateral Temporal Lobectomy in the Rhesus Monkey, with Special Reference to Psychic Blindness", *ibidem*, Volume V, page 33.

——— (1939), "Preliminary Analysis of Functions of the Temporal Lobes in Monkeys", *Archives of Neurology and Psychiatry*, Volume XLII, page 979.

Kolb, L. C. (1950), "Observations on the Somatic Sensory Extinction Phenomenon and the Body Scheme after Unilateral Resection of the Posterior Central Gyrus", *Transactions of the American Neurological Association*, page 138.

L'hermitte, J. (1939), *"L'image de notre corps"*, Paris.

——— (1951), "Visual Hallucination of the Self", *The British Medical Journal*, Volume I, page 431.

Marquis, D. G. (1934), "Effects of Removal of Visual Cortex in Mammals, with Observations on Retention of Light Discrimination in Dogs", *Association for Research in Nervous and Mental Disease, Proceedings*, Volume XIII, page 558.

——— (1935), "Phylogenetic Interpretation of Functions of Visual Cortex", *Archives of Neurology and Psychiatry*, Volume XXXIII, page 807.

Marquis, D. G., and Hilgard, E. R. (1936), "Conditioned Lid Responses to Light in Dogs after Removal of the Visual Cortex", *Journal of Comparative Psychology*, Volume XXII, page 157.

Poljak, S. A. (1933). "Contribution to Cerebral Representation of Retina", *Journal of Comparative Neurology*, Volume LVII, page 541.

Riddoch, G. (1917), "Dissociation of Visual Perceptions due to Occipital Injuries, with Especial Reference to Appreciation of Movement", *Brain*, Volume XL, page 15.

——— (1935), "Visual Disorientation in Homonymous Half-fields", *ibidem*, Volume LVIII, page 376.

Rubinstein, L. H. (1941), "Imperception for Position of Eyelids on One Side", *Journal of Neurology, Neurosurgery and Psychiatry*, Volume IV, page 191.

Russell, W. Ritchie (1948), "Functions of Frontal Lobes", *The Lancet*, Volume I, page 356.

——— (1951), Discussion on Parietal Lobe Syndromes, *Proceedings of the Royal Society of Medicine*, Volume XLIV, page 341.

Sachs, E. (1931), "The Diagnosis and Treatment of Brain Tumours", The C. V. Mosby Company, St. Louis, page 239.

Schilder, P. (1934), "Localization of Function in the Cerebral Cortex", *Association for Research in Nervous and Mental Disease, Proceedings*, Volume XIII, page 466.

——— (1935), "The Image and Appearance of the Human Body", London, Kegan Paul. Reprinted 1950, International Universities Press, New York.

Schilder, P., and Stengel, E. (1931), "Asymbolia for Pain", *Archives of Neurology and Psychiatry*, Volume XXV, page 598.

Sjoqvist, O., and Weinstein, E. A. (1942), "Effect of Section of Medical Lemniscus on Proprioceptive Functions in Chimpanzees and Monkeys", *Journal of Neurophysiology*, Volume V, page 69.

Snider, R. S. (1945), "A Tectocerebellar Pathway", *The Anatomical Record*, Volume XCI, page 299.

——— (1950), "Recent Contributions to the Anatomy and Physiology of the Cerebellum", *Archives of Neurology and Psychiatry*, Volume LXIV, page 196.

Snider, R. S., and Barnard, J. W. (1949), "Electro-Anatomical Studies on the Afferent Projection to the Inferior Olive", *Journal of Comparative Neurology*, Volume XCI, page 243.

Snider, R. S., and Stowell, A. (1942*a*), "Evidence of Tactile Sensibility in the Cerebellum of the Cat", *Federation Proceedings*, Volume I, page 82.

——— (1942*b*), "Evidence of a Projection of the Optic System to the Cerebellum", *The Anatomical Record*, Volume LXXXII, page 448.

——— (1944*a*), "Receiving Areas of Tactile, Auditory, and Visual Systems in Cerebellum", *Journal of Neurophysiology*, Volume VII, page 331.

——— (1944*b*), "Electro-Anatomical Studies on a Tactile System in the Cerebellum of Monkey (Macaca mulatta)", *The Anatomical Record*, Volume LXXXVIII, page 457.

Spiegel, E. A., and Sommer, I. (1944), "Neurology of the Eye, Ear, Nose and Throat", New York, Grune and Stratton.

Stowell, A., and Snider, R. S. (1942), "Evidence of a Representation of Auditory Sensibility in the Cerebellum of the Cat", *Federation Proceedings*, Volume I, page 84.

Turner, O. A. (1950), "Some Data Concerning the Growth and Development of the Cerebral Cortex in Man", *Archives of Neurology and Psychiatry*, Volume LXIV, page 378.

Wilson, S. A. Kinnier (1940), "Neurology", two volumes, London, Edward Arnold, page 1242.

Addendum.

The following information from Dr. J. H. Barnes, of Thursday Island, was received after this report had been completed.

The patient was admitted to hospital on July 30, 1949, very intoxicated, having fallen eight feet down stairs. It was not known whether he was unconscious after the fall. When first examined he was confused; but this was thought to be due to his alcoholic state. There was no evidence of head injury. Next morning he was confused and slept all day. Twenty-four hours after the fall he climbed out of bed and made animal noises, but returned to bed on request. Next day he was normal (the patient was always very slow—he was an alcoholic). However, his pulse rate had fallen from 76 to 60 per minute and his temperature was 100° F.

Forty hours after the fall his pulse rate was 48 per minute. The right pupil was larger than the left, there was slight paresis on the right side of the mouth, speech was slurred, and he was unable to compose a sentence, though obviously knowing what he wished to say. He was unable to protrude his tongue. His reflexes were normal, he had no headache, and X-ray examination revealed no abnormality.

During the next twenty-four hours his condition deteriorated; he was hard to arouse and had paresis of his right arm and leg. He was given 100 millilitres of 50% glucose solution intravenously and from then he began to improve mentally and physically.

One week later he could speak phrases of four to five words, slowly. He said that he was quite clear on what he intended to say; but (*a*) half-way through he would find himself saying something quite alien to his intention; (*b*) he would get the wrong word—for example, "sinus" for "sign", "morning" for "night"; (*c*) he would forget what section of his thoughts he had already expressed. He would become aware of and irritated by his mistakes, and then he would be completely aphasic.

At the end of the third week the paresis had almost disappeared and his speech was stilted but improving. Then he noted that he could not see with either eye to the right of the nasal line. He tried to read, but found it difficult; he had to call his letters aloud, and by the time he had read half a line he had forgotten the first few words. He could follow spoken sentences if they were not too long.

One month later no further progress appeared to have been made; but the patient appeared as bright mentally as he had ever been.

The original diagnosis was subdural hæmatoma; but later he was thought to be suffering from intracerebral petechial hæmorrhage.

SOCIAL AND TROPICAL MEDICINE IN THE UNIVERSITY OF QUEENSLAND.

By E. S. MEYERS,

Dean of the Faculty of Medicine, University of Queensland, Brisbane.

THE importance of social medicine is becoming increasingly recognized and students of the new curriculum of the Faculty of Medicine in the University of Queensland have just completed their year's studies in this department. Tropical medicine is of concern to Queensland practitioners, and this subject is taken in the fourth year, as well as social medicine.

The course is taken after the students have completed their study of pathology and bacteriology, so that they have a good foundation on which to base their future studies. At the same time they commence their work in clinical medicine and clinical surgery, tutorial work in these subjects having been taken in the third year. They are thus simultaneously introduced to the study of health and disease. As has been said, "a knowledge of disease constitutes only one side of a coin, the obverse of which is health and life".

A lecture is given on the evolution and the scope of social medicine, and a similar lecture is given on tropical medicine. There is a series of lectures given at 8.30 o'clock on three mornings a week throughout the four clinical terms of the year. In the fifth year the work continues for one term with lectures on forensic medicine. On Saturday mornings there is a series of demonstrations on sanitation, quarantine, processing of food, purification of water *et cetera*. For purposes of these demonstrations the class is divided into two, each half of the class spending about one term on the work. Laboratory work is carried out one day a week for three terms for a period of two hours.

The subject matter of the course is divided into six subdivisions: (i) The prevention of disease by sanitation of environment. (ii) The general principles of prevention. (iii) Social pædiatrics, which includes lectures on infant welfare, pre-school child health and the health of the school child. The period from birth to the age of about five years is divided into four stages; the first stage includes birth and the first month of life, the second stage extends from one to six months, the third stage from six months to two years, and the fourth stage from two to five years. Various questions of growth, nutrition and mental development receive special attention. (iv) Industrial hygiene: this course of lectures deals with occupation and health. (v) Tropical medicine and parasitology. Two terms are given over to practical work in parasitology, particular attention being paid to the various organisms that cause disease, and one term is given over to entomology, in which the mosquito and the fly are studied. (vi) The final section of the course is taken in the fifth year, which deals with forensic medicine. This includes lectures on identification, sudden death, electric shock, blood stains *et cetera*, legal aspects of forensic medicine, poisons and insanity, and the legal obligation of the medical practitioner associated therewith.

Social Medicine.

The following lectures in social medicine are given. The course consists of 54 lectures in social medicine and 20 lectures in forensic medicine. The subjects are as follows: (i) The evolution of the teaching of social medicine. (ii) Environment and disease: (*a*) water, (*b*) light, (*c*) food, (*d*) sanitation, (*e*) temperature, heat and cold, (*f*) air and ventilation, (*g*) housing. (iii) Industrial health. (iv) Rehabilitation, and the role of the social worker. (v) Vital statistics. (vi) Prevention of disease and immunization. (vii) Venereal disease as a community risk. (viii) Tuberculosis from an epidemiological point of view. (ix) Maternal and child welfare. (x) School health. (xi) The assessment of disability. (xii) Forensic medicine: (*a*) death, (*b*) identification, (*c*) sudden death, (*d*) wounds (I and II), (*e*) electric shock, (*f*) blood stains, (*g*) asphyxia (I and II), (*h*) sex offences (I and II), (*i*) alcoholism, (*j*) certain legal aspects (I and II), (*k*) poisons (I, II, III and IV), (*l*) insanity (I and II).

Tropical Medicine.

In tropical medicine the total number of lectures given is 45, and the subjects are as follows: malaria, hæmoflagellates, intestinal protozoa, the dysenteries, dengue and sandfly fever, venomous animals, animals as reservoirs of infection, helminths, trematodes, tropical dietary and deficiency diseases, tropical ophthalmology, cestodes, leprosy, nematodes, typhus, classical murine typhus, tick and "Q" fever, scrub typhus, leptospiroses, diagnosis of fevers, tropical ulcer, vaccination, smallpox, plague, cholera and yellow fever, quarantine, epidemiology and venereal disease.

Demonstrations.

The following special demonstrations are given.

In social medicine the subjects covered are: (i) various methods of disposal of waste: (*a*) sewage (two demonstrations), (*b*) incineration, (*c*) land reclamation; (ii) water purification; (iii) food purification (two demonstrations).

In tropical medicine a visit is made to the quarantine station. Then, on each Tuesday throughout two terms, there is a two-hour demonstration period in parasitology. Details of the work are as follows: First week, demonstra-

tion of Plasmodium; staining technique on Trypanosoma in rat's blood. Second week, staining technique in malarial and Trypanosoma blood. Third week, diagnosis of types of malaria from prepared slides; diagnosis of known malarial slides. Fourth week, continuation of same work; preparation of further slides of malarial blood. Fifth week, preparation and examination of *Entamœba histolytica* with the use of living culture. Sixth week, preparation and examination of formalized preparations of *Entamœba coli*. Seventh week, demonstration of intestinal protozoa; examination of formalized material. Eighth week, examination of fæces for *Entamœba muris*; examination of fixed slides for intestinal protozoa. Ninth week, examination of fæces for intestinal protozoa, Giardia and Trichomonas. Tenth week, demonstration of ciliates and flagellates and revision of all intestinal protozoa. Eleventh week, revision of Plasmodia. Twelfth week, demonstration of material from abattoirs (Trematoda); examination of prepared slides. Thirteenth week, Cestoda, demonstration of malarial obtained from abattoirs and prepared slides. Fourteenth week, Trematoda and Cestoda, macroscopic specimens and amounts. Fifteenth week, Nematoda, demonstration of species; examination of fresh material and prepared slides. Sixteenth week, salt flotation methods for nematodes. Seventeenth week, salt flotation methods for helminths generally. Eighteenth week, revision of all work.

Each Tuesday morning for one term there is a three-hour lecture-demonstration period in entomology. Details of the work are as follows. Lectures and practical work are conducted in the Culicidæ, the Simuliidæ, the Muscoidea, the Cimicidæ, and the Myriapoda.

An examination is held in social medicine at the end of the fourth year, and one in forensic medicine at the beginning of the second term of the fifth year.

Essays.

Students write two essays, one on the scope of social medicine in general practice, and another on a subject of their own choosing, dealing with some project associated with social or tropical medicine. The project and the second essay may be combined. The first essay is handed in at the end of the year.

The Project Method of Teaching.

The project method of teaching is used in social and tropical medicine. The second essay is handed in after the completion of the vacation at the end of the year.

The only other places where this method of teaching is used, so far as is known, are the University of Otago and the University of Malaya (Macfarlane, 1950).

The following are some of the projects carried out by the students in the vacation period at the end of the year. Essay: "The Care of the Pre-school and School Child." Project: "Investigations into the Facilities Available for the Care of the Pre-school and School Child in Brisbane." Combined: "Chemotherapy in the Eradication of Malaria." Combined: "Dietary Management at the Brisbane General Hospital." Essay: "Sociological Problem of Senility." Project: "The Marchant Home." Essay: "Epidemiological Aspects of Poliomyelitis with Particular Reference to Queensland." Project: "Investigation into the Facilities which Exist for the Care and Training of Crippled Children in Queensland." Combined: "The Incidence of Interdigital Tinea in a Group of Students from the University of Queensland." Essay: "The Medical Hazards of Bathing." Project: "Control of Swimming Pools with Special Reference to those under the Control of the Brisbane City Council." Essay: "School Health." Project: "An Investigation into Some Aspects of School Health in Queensland and Conclusions Drawn Therefrom." Essay: "The Disposal of Wastes." Project: "The Disposal of Wastes, with Particular Reference to the Difficulties Experienced in a Country Town." Combined: "Public Health in a Forestry Camp." Combined: "Accidents in Industry." Combined: "Handicapped Children." Combined: "Mosquito Prevention and Destruction." Combined: "Social Services—Do They Pay Dividends?"

Practical Experience in Social Medicine in the Various Activities in Brisbane.

In the month of October the students spend one month in seeing something of the practice of social medicine. They are divided into groups of not more than six and visit various institutions in turn. The following are the places they visit. (i) Infant health: Valley Clinic, Woolloongabba Clinic and Herschell Street Clinic. (ii) Child health: State Wooloowin Home and Lady Gowrie Centre, and School for Retarded Children. (iii) School health: Stafford State School and Kelvin Grove State School. (iv) Assessment of disability: Commonwealth Health Department. (vi) Industrial health: Ipswich workshops. (vii) Blood transfusion service: Red Cross House. (viii) Auditory and visual aids: Blind Institution. (ix) Rehabilitation centre: Perry Park.

Without the willing cooperation of the officers of the Commonwealth Health Department and the Queensland State Health Department, many of whom are lecturers in the department, it would not be possible to carry out this work.

Research.

A laboratory has been set up in the Medical School, and a commencement has been made in the collection of material for class work and research, and plans are under way for the enlargement of the museum. Diagrams and charts are being prepared. Work is only just commencing and will be extended as more staff becomes available.

Reports of Cases.

A CASE OF WATERHOUSE-FRIDERICHSEN SYNDROME WITH RECOVERY FOLLOWING THE USE OF CORTISONE, AQUEOUS ADRENAL CORTICAL EXTRACT AND CHEMOTHERAPY.

By H. P. B. HARVEY,

Royal Prince Alfred Hospital, Sydney.

MANY cases of fulminating meningococcal septicæmia, often called the Waterhouse-Friderichsen syndrome, have now been reported, with almost invariably fatal outcome. In the following case recovery followed vigorous treatment on routine lines with the addition of cortisone, the use of which has been suggested for this condition but not previously recorded.

Clinical Record.

F.S., aged seven years, was sent home from school early on February 13, 1951, because she felt ill, had a headache and had vomited once. At home she vomited again and complained to her parents of a severe headache. She was brought to the casualty department at the Royal Prince Alfred Hospital, where she was found to have enlarged and inflamed tonsils and a temperature of 102° F., but no other localizing sign of infection.

During the night the child vomited many times and was very restless. By morning she was confused and lethargic, with a blotchy rash on her legs, and appeared alarmingly ill. She was brought back to the Royal Prince Alfred Hospital, where she was immediately admitted with the diagnosis of "meningococcal septicæmia".

The child seemed moribund. Closely grouped over her lower limbs and buttocks, extending as high as the iliac crests, was a striking collection of petechial hæmorrhages of varying size up to one and a half inches in diameter. In the next few hours many more of these ecchymoses appeared on the legs and buttocks, and several now appeared on the shoulders and one on the left elbow.

She was semi-comatose, with eyes half-open and rotated under the upper lids, awakening only to protest feebly at the minor surgical procedure which was performed, and

then lapsing back into unconsciousness. Her respirations were irregular and shallow, and numbered 10 per minute. Her skin was deathly pale and cold, and there was a cyanotic hue of the lips, lobes of the ears and extremities giving the typical cadaveric appearance often described in this condition.

No pulse could be felt at the wrist, and the first estimation of her blood pressure was 25 millimetres of mercury, systolic, and 10 millimetres, diastolic. Immediately afterwards other observers could not obtain any values. She had pronounced neck rigidity and Kernig's sign was present. Examination of the heart and lungs revealed no further abnormality, and no masses were palpable in her abdomen.

Immediately on her arrival in the ward the left saphenous vein was exposed at the ankle and a cannula was inserted. Blood taken from this later yielded meningococci in pure culture. Ten millilitres of "Eschatin", 200,000 units of penicillin and two grammes of soluble sulphadiazine were injected consecutively, and a flask containing 600 millilitres of 4% glucose solution in N/5 saline, with one gramme of soluble sulphadiazine and 10 millilitres of "Eschatin" added, was connected to the cannula, the fluid being allowed to run into the vein during the next forty minutes. Similar quantities of fluid with the same composition were then given every eight hours for forty-eight hours.

At the same time a lumbar puncture was performed, some turbid fluid was removed and 20,000 units of dilute penicillin solution were injected. The fluid collected had a chloride content of 690 milligrammes *per centum*, a sugar content of 17 milligrammes *per centum*, and a protein content of 500 milligrammes *per centum*, and later yielded a growth of meningococci in pure culture; it contained 4000 leucocytes per cubic millimetre, 95% being polymorphonuclear cells. Subsequent lumbar punctures were performed at intervals of twenty-four and forty-eight hours, and 20,000 units of penicillin were injected. The fluid was still purulent, but was now sterile.

Penicillin, 1,000,000 units, was given by intramuscular injection immediately and thereafter every two hours for forty-eight hours. Cortisone acetate, 100 milligrammes, was given intramuscularly at the outset, the dose being repeated at twenty-four and forty-eight hours. Then 50 milligrammes were given daily for a further two days.

The only other investigation was a blood count on her admission to hospital, which gave the following results: the hæmoglobin value was 13·3 grammes *per centum*; the leucocytes numbered 18,850 per cubic millimetre, 94% being neutrophile cells (a marked "shift to the left" being present), 4% lymphocytes and 1% monocytes; no eosinophile cells were seen; the platelets numbered 46,500 per cubic millimetre.

The primary therapy was completed within twenty minutes and the response was instantaneous and dramatic. The patient's blood pressure rose in ten minutes to 90 millimetres of mercury, systolic, and 50 millimetres, diastolic, and two hours later the figures were 105 and 90 millimetres respectively. The cyanosed cold extremities rapidly became pink and warm, and the pulse became easily palpable at the wrist. She became more restless, and her respirations became deeper and regular at a rate of forty per minute, and soon afterwards she lapsed into sound sleep, from which no attempt was made to arouse her.

Meningitis now dominated the picture. Neck rigidity and opisthotonus with extreme photophobia and irritability were outstanding. She maintained her peripheral circulatory recovery of the first ten minutes, and her temperature and respiratory rate were normal at the end of seventy-two hours. Her pulse rate decreased more slowly and was not down to a basic level for two weeks. She did not vomit, appeared adequately hydrated and passed urine freely into the bed. By the end of the third day she was convalescent.

Fluids were given by mouth as well as by a very slow intravenous "drip", and though some vomiting occurred in the next two days, by the fifth day she was being given a light diet by mouth and the intravenous administration of fluids was suspended. "Eschatin" therapy was suspended after three days. On the fourth and fifth days 50 milligrammes of cortisone were given, then it too was suspended. The intravenous administration of sulphadiazine was suspended at seventy-two hours, but it was given orally for a further three days. The intrathecal administration of penicillin was suspended after the third day, but the intramuscular administration was continued, 500,000 units being given every four hours for three days, then every eight hours for a further three weeks to counter secondary infection of the widespread sloughing areas of skin.

By the fifth day after her admission to hospital the child was sitting in her cot and playing with toys. She spent a further eight weeks in hospital while the sloughing and necrotic areas of skin overlying the original ecchymoses granulated and epithelialized. Skin grafting was considered unwarranted by the consultant plastic surgeon, and the areas were treated as though they were second-degree burns. On her discharge from hospital she was a perfectly well child in every respect, apart from the widespread scars on her legs and buttocks.

Commentary.

Since Waterhouse (1911) and Friderichsen (1918) first described the syndrome with which their names have been linked, many hundreds of cases have been collected in the literature.

This syndrome is usually characterized by gross purpura, collapse and peripheral circulatory failure, respiratory failure, cyanosis and rapid death. The importance of the damage to the adrenal cortex has not been decided. That gross adrenal destruction by hæmorrhages is not invariably present has been shown by Daniels (1948), who found these absent in 10% of a series of 300 autopsies on subjects with Waterhouse-Friderichsen syndrome.

The fact that patients dying from other types of overwhelming infection and from extensive burns may suffer bilateral adrenal hæmorrhage led Carey (1940), Morison (1943), Daniels (1948) and others to consider that the adrenal hæmorrhages might be secondary agonal phenomena and not the cause of the sudden circulatory collapse. Moreover, an adrenalectomized dog will live for some days, whereas a person with overwhelming meningococcal septicæmia may succumb in several hours. Also Carey (1940), Daniels (1948), Isaacson (1947), Holmes and many others have reported cases in which recovery occurred without the aid of adrenal cortical extract.

That chemotherapy is essential is well demonstrated by the fact that no authenticated recovery has occurred without the use of at least the sulphonamide drugs. However, Rich (1944) considers that adrenal insufficiency plays a major part in the syndrome and has been able to demonstrate specific adrenal lesions in these cases quite different from those occurring in other types of shock. It must also be remembered that an adrenalectomized animal will rapidly succumb to an infection which a healthy animal would easily overcome.

Banks (1942, 1943) and many others have stated the need for adrenal cortical extracts in this as well as in other overwhelming infections.

In the case presented the immediate dramatic response to therapy could not be attributed to a sudden victory for the chemotherapeutic agents and the instantaneous removal of the bacterial toxins from the system, but could have been due to the support given to a failing adrenal cortex which, as Kneeland (1951) states, had received an "acute insult".

Summary.

1. A case of the Waterhouse-Friderichsen syndrome with recovery following the use of chemotherapy, intravenous fluid administration and adrenal replacement therapy with "Eschatin" and cortisone is presented.

2. The need for adrenal cortical replacement therapy is discussed.

Acknowledgement.

I wish to thank Dr. A. W. Morrow for permission to report this case and for his guidance in its preparation.

References.

Banks, H. S., and McCartney, J. E. (1941), "Meningococcal Adrenal Syndromes and Lesions", *The Lancet*, Volume I, page 219.

Carey, T. N. (1940), "Adrenal Hemorrhage with Purpura and Septicemia (Waterhouse-Friderichsen Syndrome) with Recovery", *Annals of Internal Medicine*, Volume XIII, page 1740.

Daniels, W. B. (1948), "Meningococcic Bacteremia", *Archives of Internal Medicine*, Volume LXXXI, page 145.

Friderichsen, C. (1917), "Suprarenal Hæmorrhage in Infants", *Ugeskrift for læger*, Volume LXXIX, page 1817.

Isaacson, I. (1947), "Fulminating Meningococcal Septicæmia; Report of a Case Showing Organisms in Direct Blood Smear; Recovery", *Clinical Proceedings*, Volume VI, page 71.

Kneeland, Y. (1951), "Meningitis", *The American Journal of Medicine*, Volume X, page 739.

Morison, J. E. (1943), "Bilateral Adrenal Hæmorrhage", *The Lancet*, Volume I, page 801.

Waterhouse, R. (1911), "A Case of Suprarenal Apoplexy", *The Lancet*, Volume I, page 577.

Reviews.

SKULL FRACTURES AND BRAIN INJURIES.

A LARGE and expensive volume by H. E. Mock[1] contains an unusual presentation of his experience in 1100 cases of head injury seen during the past twenty-five years, together with an analysis of the hospital records of 6000 additional cases in which treatment was carried out in a group of hospitals over the same period.

In an early chapter the author gives the results of treatment under conditions obtaining at the present time. The mortality rates in various hospitals are reviewed and compared with the standard of treatment given. The results of this survey are salutary, and the effect is further enhanced by a series of case reports which describe in detail some of the mismanagement to which patients with head injuries are still subjected. The author states that the survey of material from the group of hospitals shows that in all clinics cases of flagrant mismanagement occur, and the reader feels that the time, energy and money put into this large monograph would be more than justified if this appalling state of affairs could be diminished.

Following this stimulating beginning the author sets out his "Common Sense Principles and Rules" for the management of head injuries. Once again, each point is stressed by examples of mistakes in diagnosis and management which leave one with many misgivings concerning the professional ability of some practitioners.

From this point the book becomes more laboured as more case histories are reproduced in detail. Some of this information is relevant, some irrelevant but nevertheless interesting, but much is both irrelevant and uninteresting. Many pages are burdened with anatomical detail which most readers will skip. This unnecessary padding is one of the serious defects of the book.

The phrasing of the text is poor and often adds to the difficulty in reading. One finds in italics that "subarachnoid hæmorrhage is a symptom and not a disease entity", but it takes some time to realize from the following paragraph that the author means that such a hæmorrhage is more frequently a manifestation of a cortical laceration than of hæmorrhage from vessels in the subarachnoid space.

A book of 800 pages on such a subject as this is bound to provoke a considerable amount of controversial comment, and one is prepared to find many personal viewpoints set out by the author which will not receive universal acceptance, but most readers will find many conceptions too narrow and uncritical.

Throughout the book the author draws freely upon the statistical analysis of over 6000 hospital records. In much of the text these statistics are used to strengthen certain opinions and they do add to the general clinical picture which the author is endeavouring to construct. But in discussing prognosis the author classifies the serious prognostic signs and gives the mortality associated with each in an unrelated fashion which may be misleading; for example, it is suggested that if a patient is incontinent he has only a 50% chance of survival.

The author is a well-known advocate of spinal drainage and he presents this form of therapy as the most generally beneficial measure in the routine treatment of head injuries. A wide range of indications are given for spinal drainage, but there is not sufficient information about its limitations, nor is the danger of its uncritical use sufficiently stressed.

In spite of all the numerous defects of the book it remains of interest because it does represent the development of thought upon head injuries over a quarter of a century. Although the author may be criticized, he has given much thought and enthusiasm to his hobby, and the honest interpretation of his observations must make many surgeons think more about head injuries. With more careful and perhaps ruthless editing a good monograph with a wide appeal would have been obtained and a real case for spinal drainage would have been presented. In its present form useful and relevant information is overburdened by unnecessary padding and many uncritical statements.

[1] "Skull Fractures and Brain Injuries", by Harry E. Mock, M.D.; 1950. Baltimore: The Williams and Wilkins Company. Sydney: Angus and Robertson, Limited. 9½" × 6½", pp. 820, with 116 illustrations. Price: £7 5s. 3d.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Modern Medication of the Ear, Nose and Throat", by Noah D. Fabricant, M.D., M.S., with a foreword by Austin Smith, M.D.; 1951. New York: Grune and Stratton. 9" × 6", pp. 262, with 20 illustrations. Price: $5.75.

The book is intended "to meet the day-by-day requirements of physicians interested in the practical application of medication of the ear, nose and throat".

"An Introduction to Medicine for Nurses", by Patria Asher, M.D., M.R.C.P., with a chapter on Mental Ill-Health by Portia Holman, M.A., M.R.C.P., D.P.M.; Second Edition; 1951. London: William Heinemann (Medical Books), Limited. 9" × 6", pp. 376, with 93 illustrations. Price: 25s.

The object is to provide nurses with a complete text-book suited to their requirements.

"Atlas of Genito-Urinary Surgery", by Philip R. Roen, M.D., F.A.C.S., with an introduction by Clarence G. Bandler, M.D., F.A.C.S.; 1951. New York: Appleton-Century-Crofts, Incorporated. 10" × 7", pp. 338, with many illustrations. Price: $8.00.

The object of the book is to present line drawings of the step-by-step progression of operative genito-urinary procedures.

"The Care of the Ageing and Chronic Sick", by A. P. Thomson, M.C., M.D., Ch.B. (Birmingham), F.R.C.P. (London), C. R. Lowe, M.D., Ch.B. (Birmingham), M.R.C.S. (England), L.R.C.P. (London), D.P.H., and Thomas McKeown, B.A. (British Columbia), Ph.D. (McGill), D.Phil. (Oxford), M.D. (Birmingham), M.B., B.S. (London); 1951. Edinburgh: E. and S. Livingstone, Limited. 10" × 7", pp. 134, with 18 text figures. Price: 7s. 6d.

Consists of articles reprinted from the *British Medical Journal* and the *British Journal of Social Medicine* and of a discussion from *Proceedings of the Royal Society of Medicine*.

"Food and Nutrition", by E. W. H. Cruickshank, M.D. (Aberdeen), D.Sc. (London), Ph.D. (Cantab.), M.R.C.P.; Second Edition; 1951. Edinburgh: E. and S. Livingstone, Limited. 8½" × 6", pp. 454, with 51 illustrations. Price: 30s.

The first edition was published in 1946.

"Chiropodial Orthopædics", by Franklin Charlesworth, F.Ch.S., with a foreword by Harry Platt, M.S., F.R.C.S., F.A.C.S.; 1951. Edinburgh: E. and S. Livingstone, Limited. 9" × 6", pp. 264, with 131 illustrations. Price: 25s.

Deals with apparatus for the relief of discomfort and disablement caused by deformities of the feet.

The Medical Journal of Australia

SATURDAY, FEBRUARY 16, 1952.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: surname of author, initials of author, year, full title of article, name of journal without abbreviation, volume, number of first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

THE DEATH OF HIS MAJESTY KING GEORGE VI.

His Majesty King George VI has died in his sleep and his people stand in silent reverence at the news. Called suddenly to his high office, he showed through the years by his deeds and his way of living that he was a man of rare understanding and high purpose, of sincerity and courage. He called forth loyalty in the hearts of his people and inspired in them genuine affection which bound them closely to him. That he recovered from the stress of his recent illness and its heroic treatment gave hopes that at least some years of serenity would be granted to him. but it was not to be, and he had to lay his burden aside. The members of the medical profession join with the whole people of the Commonwealth in appreciation of the life and character of His late Majesty, in sympathy with Her Majesty the Queen Mother, Queen Elizabeth II and the Royal Family, and in constant and direct loyalty to the Throne.

EVEN CHILDREN ARE PEOPLE.

MANY adults, if we may judge by what they do, hold a curious theory about small children. It is that the child neither sees nor hears what is going on round about, unless the adult wills otherwise, or that if he does hear or see something he does not understand it or care about it. The story is told of an exceptionally handsome small boy whose good looks were, not for the first time, being extolled in his presence by a female visitor. He capped the gushing comment with the remark: "Most people admire my eyelashes as well." If the adults present were astonished, they had no right to be. An extension of the adult theory is that children have no finer feelings or opinions that warrant consideration, and that if adult requirements or uncontrollable circumstances dictate a certain course of action, such as a city shopping expedition or a visit to the doctor, the child should acquiesce in the manner of an extra piece of luggage and require no explanation. Then if the child responds with tears to boredom or physical weariness, he is naughty; or if he objects to unexpected hurt or indignity, or shows terror at being unexpectedly put into hospital, he is unreasonable. The fashion of the completely uninhibited child has largely had its day, and few will lament its passing; life just is not like that, and it is well for the child to learn that "no man liveth unto himself". Nevertheless, the child has a point of view, which if not allowed reasonable expression may well show itself in other ways even less convenient. It is true, of course, that the adult who fails to make allowances for the child's point of view does not always mean to be unkind—quite the contrary in many cases. Too often the situation is that described by the youthful narrator in Kenneth Graham's book "The Golden Age" (speaking of his aunt and uncle guardians):

> They treated us, indeed, with kindness enough as to the needs of the flesh, but after that with indifference (an indifference, as I recognize, the result of a certain stupidity), and therewith the commonplace conviction that your child is merely animal.

There is the paradox. The charge made by the child against the adult is stupidity.

The attitude of the child towards doctors and hospitals can be tremendously important. A significant example is the damage done by bungling when the child is admitted to hospital. The following situation, which he tries to avoid in his own hospital, has been described by the senior medical administrator of one of our larger children's hospitals. The child is brought to the hospital either for prearranged admission or for advice about something that is found to necessitate admission. To simplify the visit in the first place the mother has either said nothing or told the child that they will come straight home after going to hospital, or has made some other evasive or false statement—very often in a mistaken attempt to be kind and sometimes half-hoping that it may be true after all. At the hospital the doctor says little to the child—little, at any rate, that the child regards as important. Then, with no real explanation, the child finds himself separated from his mother, taken away by a nurse and in a strange frightening place. His world is in ruins. The foundations of his security have turned to sand. His mother is a liar, the doctor is an ogre, and the hospital is a prison, no doubt complete with torture chamber. This is not an overdrawn picture as it exists for many children, and it unquestionably leaves a permanent mark. It is an indictment of adult stupidity that can be avoided. We cannot do much about what the parent says, but what the admitting doctor says and does may make all the difference to the child's attitude. The child is entitled to some statement and attempt at explanation by the doctor, and even if reconciliation to admission to hospital is not possible, at least the doctor can accept the responsibility and leave undamaged the child's confidence in his mother with all that it means to a basic sense of security. Incidentally,

it seems fair to comment that the making of an explanation intelligible and acceptable to the child is a test of the doctor's intelligence as much as of the child's.

An even more striking picture, that is painfully familiar, has been drawn by an American psychiatrist, A. Z. Barhash,[1] on the subject of tonsillectomies in children. Barhash deplores the fact that many doctors fail to share much information with their patients about either diagnosis or treatment and points out that it is hardly surprising that they share even less with children. They assume that children would not understand anyway; and if they did understand, the information would only make them less tractable. They count on a safe outcome, knowing that the surgical risk is small, but fail to realize the emotional trauma that can be inflicted. To the doctor tonsillectomy is "nothing more than a minor operation in which the two most frequent annoyances are an occasional bleeder or a loudly screaming child". To the child it is much more, especially if he is unprepared or falsely prepared with an evasion or a lie. First he is taken from home hungry and brought to a strange and somewhat fearsome place. His apprehension, initiated by hunger, increases as he waits, often for long periods, and sees and hears strange things. He is then separated from his mother, often by a strange and sometimes masked attendant or nurse. He sees other masked and robed figures, he smells strange smells, is given a painful injection, has a mask put over his mouth and nose, and is forced to breathe something which chokes him. Is it too surprising, Barhash asks, that to the uninitiated child the most frequent conclusion is that he is being killed, and that all the strange doings are nothing more than an accompanying ritual? He awakens ill and with a sore throat that suggests that he has been stabbed in the throat. He is usually still among strangers and probably sees his mother only when he has recovered from the anæsthesia, if then. Though he has escaped death, the anxiety which accompanies the miraculous escape stays with him, as do feelings of confusion and resentment. The picture is not, of course, true in detail always and everywhere, but we have all seen enough of it to be provoked to thought. Barhash's comments are constructive. He urges that the doctor be honest with the child, and informative rather than secretive. The child, according to his age and level of understanding, should be told what is wrong, what the anatomical facts are, what is going to be removed, how it is going to be removed, and what the effects are. To the bland assumption that children understand nothing and therefore no explanation is needed, Barshak replies that those who work among children realize that they understand much more of the important things than they are given credit for. What they fail to understand are not facts and ideas, but rather our complex and garbled terminology. Another important point is to try to overcome the aspect of strangeness associated with a first operation. If possible the child should see something of the hospital, if not of the operating theatre, in advance, and should smell and handle, perhaps have an opportunity to play with, an ether mask. A doctor or nurse whom the child knows should take him from his mother, waiting about should be eliminated as far as possible, and the mother should be with the child to the point when he is anæsthetized and on his awakening. The doctor should make himself aware of possible sources of anxiety for the child and should try to anticipate them. This all sounds like a fearful lot of bother, and some people will regard it as nonsense. It is as well to remember that comments of the same sort have been made before, for example, about Semmelweis's and later Lister's views on antisepsis. Also, incredible as it may seem today, many commented bitterly on the reduction of working hours and improvement of conditions of work of children in factories early in the nineteenth century.

Without a doubt like comments will be made about an experiment being carried out at the Hospital for Sick Children, Great Ormond Street, London, though it too is based on the idea that the child in hospital is a human being. Most of our readers will be able to examine for themselves the preliminary account[1] of the experiment given by Alan Moncrieff, who is Nuffield Professor of Child Health in the University of London, and A. M. Walton, who is a ward sister at Great Ormond Street, but the main points may be mentioned. Apparently it applies more particularly to the "toddler" group. The plan is that mothers are encouraged, and even strongly urged, to visit their children in hospital daily between 5 and 6 p.m. They give their children their suppers, or feed their infants. They read a story or play a game, clean teeth, hear prayers, and tuck the children down for the night. That is, they do in hospital the things they would do for the children at home. If tears threaten at the time of parting, the mother tries to stay until the child is asleep. If a mother cannot come, other mothers act as a substitute. The child can look forward to tomorrow. The early part of the day in hospital is not unexciting, and if "Mum" is expected after tea homesickness is kept at bay. As Moncrieff and Walton put it, the toddler can understand today and tomorrow and can associate things with a meal-time. It is felt that the plan helps to continue the relationship between mother and child and does not break up the security which means so much to young children. The child's needs are satisfied jointly by mother and nursing staff without the development of abnormal possessiveness by the nursing staff or transfer of affection. The mothers are less strained because they have something to do in their visiting time, and incidentally they are encouraged to interview the medical staff, especially the visiting staff. There are difficulties associated with travelling and the needs of the other members of the family, but the plan, which has operated for eighteen months, seems to be working. According to Moncrieff and Walton, the mothers like it, the nursing staff like it, and the children seem happy. What the medical staff feel about it is not mentioned. Let us hope that they are not viewing the experiment too impatiently, and that if on prolonged trial it proves a success they will commend it to others. There is a breath of humanity about it which is surely in its favour. Children are much more impressionable than adults, and our attitude to them in hospital demands the most searching scrutiny if we are to avoid doing things to them that are in their own way as harmful as careless medical treatment or bad surgery. Not long ago we pointed out in these columns that "the patient is a person". Gratuitous as the comment will be to many, there seems ample justification for adding as a corollary that even children are people.

[1] *The Journal of the American Medical Association*, August 25, 1951.

[1] *British Medical Journal*, January 5, 1952.

Current Comment.

A POSITIVE ATTITUDE TOWARDS DISSEMINATED SCLEROSIS.

MEN and women suffering from disseminated sclerosis need before everything else courage and hope tempered by an understanding of the truth. Many medical advisers, remembering the facts as they know them and looking back on their experience, are often so depressed at the prospect of diagnosing disseminated sclerosis or undertaking the care of one of its victims that they are powerless to engender courage or hope. This feeling of depression is understandable, but the automatic adoption of a hopeless and so negative attitude is not always justified. We seem to be no nearer any form of treatment that will cure or even influence the progress of the essential disease process (this was made clear in a discussion in these columns on March 17, 1951, on related drug therapy in the light of a definitive American report), but a good deal is being done in some places to give positive assistance to those affected by disseminated sclerosis. The keynote of this, as brought out in a recent article by E. E. Gordon and K. E. Carlson,[1] is to make the most of the patient's capabilities. Disability caused by the disease is real and often distressing, but its course is by no means always progressive and swift, and the patient has to live with it. Many will be surprised to know that the median duration of the disease has been recently estimated as in the vicinity of twenty-seven years. Moreover, remissions may last for years. It is not possible in an individual case to predict the immediate course and prognosis, and in most cases the only fair approach of the medical adviser (though often not the easiest) is to be humanely frank and realistic with the patient and then to act according to the most reasonably optimistic prognosis. As Gordon and Carlson put it, "every patient must be given the benefit of the doubt to as liberal an extent as the judgement of the physician will permit". The doctor must foster a spirit of hopeful management and exploit every device to improve the patient's situation, in particular making full use of the techniques of rehabilitation that have received much attention in recent years. The most important thing is not what the patient cannot do, but what he can do. His capabilities need to be exploited to the full. The object is one of "teaching the patient how to manage his life in spite of his handicaps and by means of his residual abilities . . . of calling into play reserve functions and of developing substitution devices". Gordon and Carlson summarize the techniques used to realize these aims as follows: first, the strengthening of residual motor units of a muscle which are only partially removed from voluntary innervation, but which are atrophied from disuse; second, the training of synergists for a particular motor pattern in which the neuronal connexions to the prime mover are affected; third, the training of other muscles to perform movements to substitute for the normal prime movers irreversibly affected; fourth, the use of mechanical devices to compensate for disabilities in order to accomplish purposeful motion required for the numerous activities of daily living. We need not concern ourselves further here with Gordon and Carlson's elaboration of the training process of "neuro-muscular re-education", as they term it. It can be read by those interested, and ample references are given in their paper to sources of more detailed information. The important point is that a programme along these lines, combined with such other needs as the management of bladder disturbances and the promotion of good hygiene, both mental and physical, has produced worthwhile results when it has been conscientiously put into effect. It calls for considerable knowledge, skill and patience and may not often be within the province of the family doctor, though he could do a good deal once he realized the possibilities and could well carry on the supervision of an established programme. The immediate difficulty in most parts of Australia, if the practitioner does not feel able to deal with the situation, is to know where to direct the victim of disseminated sclerosis and other chronic neurological disorders. If there are places with adequate facilities and competent medical and physiotherapy staff, they are not well known. Excellent work is being done for many children with chronic neurological and neuro-muscular disorders. The organized orthopædic and physical medicine groups within our profession might well apply themselves to the needs of adults. If not we shall hesitate to try to add hope to the very real courage that many of these people already possess.

[1] *The Journal of the American Medical Association*, October 20, 1951.

THE EMPIRE MEDICAL ADVISORY BUREAU AND VISITORS TO THE UNITED KINGDOM.

THE arrival of the Empire Medical Advisory Bureau's summary of regulations for post-graduate diplomas and of courses of instruction in post-graduate medicine, dated December, 1951, serves as a reminder of the services offered to visitors by this bureau. Established in London by the Council of the British Medical Association, the bureau aims at welcoming and providing a personal advisory service to practitioners visiting the United Kingdom, particularly those from the Dominions and Colonies. Subjects on which service is offered include post-graduate education, whether related to higher qualifications or not, accommodation, private hospitality and general information on local circumstances, leisure activities and so on. The summary of regulations and courses provides comprehensive information on all aspects of post-graduate work. The bureau appreciates as long notice as possible of an intended visit to the United Kingdom and information on the following lines: projected date of arrival, mode of travel, whether accompanied by wife, period of stay, main and other objects of visit, and requirements from the bureau. A letter of introduction from the local Branch of the British Medical Association is helpful, but not essential. In any case visitors are invited to make contact with the bureau on arrival and to seek advice or assistance from the Medical Director. All communications should be sent to Dr. H. A. Sandiford, M.C., D.P.H., Medical Director, Empire Medical Advisory Bureau, British Medical Association House, Tavistock Square, London, W.C.1.

GENITO-CRURAL PRURITUS FROM ORAL AUREOMYCIN THERAPY.

GENITO-CRURAL pruritus can be an unpleasant side effect of oral aureomycin therapy. This may not be generally known, but A. J. Reiches and P. K. Webb[1] have reported 13 cases and it has been described previously. The genito-crural symptoms may occur with or without diarrhœa. In the series of 13 cases reported, erythema, œdema, scratch marks and, in one case, secondary infection were noticed in the involved areas. The actual cause of the symptoms is still a matter for discussion. Reiches and Webb point out that sensitization to aureomycin can and does occur, and a number of cases have been reported in the literature. They suggest that after oral administration of aureomycin, portion is unabsorbed; this, coming into contact with the anal, perianal and perineal skin, acts as a sensitizer which in all probability causes the intense burning and itching. Another point of interest is that aureomycin causes changes in the intestinal flora and appears to interfere with factors which limit the growth of *Candida albicans*. The increase in the amount of this organism may be an added irritating factor. On the basis of the sensitization theory Reiches and Webb treated their patients by the local application of an antihistamine ointment three times a day with gratifying results. Relief was obtained within twenty-four to thirty-six hours, and in one case within a few hours. Their experience was limited to phenindamine ointment, but it may be expected that other antihistamine preparations would be effective.

[1] *A.M.A. Archives of Dermatology and Syphilology*, July, 1951.

Abstracts from Medical Literature.

PATHOLOGY.

An Unusual Recurring Connective-Tissue Tumour.

W. ST. C. SYMMERS AND E. J. NANGLE (*The Journal of Pathology and Bacteriology*, July, 1951) describe four cases of an unusual recurring connective-tissue neoplasm. The tumours consisted of immature vascular, fibrous and adipose tissues resembling the embryonic connective tissues which develop from the undifferentiated mesenchyme. No evidence of hæmatopoiesis was found in any of the four cases, although cells resembling nucleated precursors of erythrocytes were seen; they were interpreted as phagocytes. In one case the tumours were frankly sarcomatous in structure; in the others they were histologically benign. This is considered to be of little importance in relation to prognosis, as local recurrence developed in all cases. The authors state that metastasis was not observed in any of the cases, although its development would not be surprising. Two of the patients were men and two were women. The ages at which the tumour was first noticed ranged from twenty-three to forty-seven years, and the durations before excision from eight weeks to three years. The intervals between the first excision and the first recurrence ranged from thirteen months to more than four years (eighteen months for the histologically sarcomatous growth). In three cases amputation was necessary thirty-five, thirty-seven and fifty-two months respectively after the tumour was first noticed. In the third case the patient died with intra-abdominal extension of the tumour little more than a year after hind-quarter amputation. In the fourth case the tumour recurred repeatedly over a period of twenty-four years despite surgical and deep X-ray therapy; the recurrent tumours were moderately radiosensitive. Treatment by amputation is advised if recurrence follows local excision. If amputation is impracticable recurrence should be treated by a combination of excision and radiotherapy.

Cylindromatous Mucous-Gland Tumours of the Trachea and Bronchi.

R. H. R. BELSEY AND J. C. VALENTINE (*The Journal of Pathology and Bacteriology*, July, 1951) describe three cases of a cylindromatous type of tumour of the trachea and bronchus, in one of which ciliated epithelium was present as part of the neoplastic process. The literature is reviewed and a similarity traced between these tumours, salivary-gland tumours and basal-cell carcinomata of the skin. It is suggested that they be classified as a subgroup of the mucous-gland tumours of the trachea and bronchi, and that they be separated from the more usual type of adenoma of the bronchus, to which they are related by a common ancestry from primitive replacement epithelium. The authors state that cylindromata of the trachea and bronchi behave as carcinomata of low-grade malignancy, with a pronounced tendency to local recurrence. They rarely metastasize, but invade locally, and are rather more malignant than the ordinary adenoma.

Suprarenal Hæmorrhage and Necrosis in Pregnancy.

MARGARET D. CRAWFORD (*The Journal of Pathology and Bacteriology*, July, 1951) reports 14 cases of hæmorrhage and necrosis of the suprarenal cortex during pregnancy and the puerperium. The histology of the lesions is described and the clinical aspects of the cases are discussed. The author states that massive infection and the toxæmias of late pregnancy associated with severe malnutrition appear to be possible ætiological factors. In non-fatal cases the patients may later develop Addison's disease and have severely scarred or almost non-existent suprarenal cortices; they presumably form part of the group previously described as having a condition of "primary contracted suprarenals" or "atrophy of the suprarenals", but more appropriately named post-necrotic scarring.

A Biopsy Study of Chronic Gastritis and Gastric Atrophy.

R. MOTTERAM (*The Journal of Pathology and Bacteriology*, July, 1951) describes the morphology of the mucosa of the body of the stomach in 150 patients suffering from a variety of dyspeptic symptoms, in whom an opaque meal examination showed no gross abnormality. He states that changes, when present, could be grouped into two broad categories: (*a*) superficial gastritis without atrophy of tubular epithelium, and (*b*) more deeply extending gastritis with atrophy of tubular epithelium. Mucosa from patients with superficial gastritis due to excess of alcohol returned to normal after withdrawal of the alcohol. In 43 cases of pernicious anæmia, complete or almost complete tubular atrophy combined with prominent intestinal metaplasia and absence of wandering-cell infiltration made up a pattern which is distinctive for this disease. Adequate liver-extract therapy failed to alter these mucosal appearances.

Metastasizing Goitre.

R. CUNLIFFE SHAW (*The British Journal of Surgery*, July, 1951) states that metastasizing goitre of the so-called Hürthle-cell type is a condition of great rarity and corresponds to the *struma post-branchialis* of Langhans and Getzowa and the parastruma of Langhans. The principal histological elements in these tumours are the water-clear cells and the finely granular large eosinophilic cells. According to some, these cells represent two phases of activity of the same epithelium, being the resting and the secreting phases. Some regard these elements as evidence of parathyroid origin, particularly if the water-clear cells predominate. On the other hand, if the granular oxyphilic cells are mostly in evidence, the tumour is considered to be of thyroid origin. There appear to be two distinct elements present in these tumours. If the thyroid element predominates it is suggested that the thyroid alveolar structure seen in the metastases of the so-called benign metastasizing goitres may be due to the slower development of the proliferative struma type of tumour, whereas in the cases in which the water-clear cells are an outstanding histological feature the tumour is rapidly developing and does not approximate to the normal thyroid, being more closely linked to the parathyroid. There is a distinct difference between the clinical course of these rare tumours classified as atypical carcinomata and the malignant tumours belonging to the well-recognized histological types. The latter tend to disseminate in over 70% of the cases to the regional nodes in the neck, whereas the former usually metastasize to bone and especially to the skull, the regional nodes being involved in only a few cases. Some of these cases show a mild degree of thyrotoxic symptoms, but these changes are not associated with either the water-clear cells or those of the proliferative struma type; they appear to arise from independent thyrotoxic changes in other portions of the thyroid gland. The clinical course of the metastasizing struma-parastruma (Hürthle-cell tumour) is singularly inoffensive until pressure symptoms arise, commonly from a remote secondary growth. The primary lesion of the thyroid develops very slowly and produces negligible clinical effects. Thyroidectomy for the primary tumour and radiotherapy for the secondary metastases would appear to be the most appropriate method of treatment.

The Adenoma-Carcinoma Sequence in Cancer of the Colon.

RAYMOND J. JACKMAN AND CHARLES W. MAYO (*Surgery, Gynecology and Obstetrics*, September, 1951) state that the concept of adenoma-carcinoma sequence in carcinoma of the large bowel is supported by (i) the close parallelism between the location of polypi and the location of carcinoma in various segments of the colon, (ii) the close parallelism in age and sex incidence, (iii) the frequency with which polypi are associated with carcinoma (the "sentinel" polypus), (iv) the frequency of malignant degeneration in familial multiple polyposis, (v) subsequent replacement of known solitary adenomata by frank carcinoma, and (vi) polypoidosis-carcinoma complications of chronic ulcerative colitis. Many adults in the age group of thirty to seventy years (variously estimated at from 7% to 17%) have polypi and in a few instances early carcinomata of the large intestine which are entirely asymptomatic.

Preclinical Paget's Disease of the Nipple.

MALCOLM B. DOCKERTY AND STUART W. HARRINGTON (*Surgery, Gynecology and Obstetrics*, September, 1951) state that preclinical Paget's disease of the nipple was originally described as a clinical entity in which an eczematoid lesion of the nipple sometimes was followed by the development of an underlying mammary carcinoma, and has reached a status in which investigators overemphasize the gross clinical features of the cutaneous component. Not all conditions in which there is excoriation of the nipple are examples of Paget's disease, but certain peculiar malignant characteristics of the involved skin fortunately are pathognomonic of true Paget's disease. In the experience of many who have studied examples of Paget's disease, these cutaneous changes always are accompanied, if not

preceded, by the development of an adenocarcinoma in the underlying breast. The "deep" carcinoma tends to be multicentric, and its origin seems to be from the lining of the large ducts. The authors present a series of seven cases of Paget's disease in which no macroscopic signs of abnormality of the nipples could be detected, the clinical picture being that of ordinary mammary carcinoma, sometimes of several years' duration. They state that evidence strongly suggests that in these cases the deep glandular carcinoma preceded in point of time the evolution of the cutaneous changes, which, although typical of Paget's disease, were found only after careful and prolonged microscopic search.

Cardio-Vascular Responses to Air Embolism.

G. R. CAMERON, S. N. DE AND A. H. SHEIKH (*The Journal of Pathology and Bacteriology*, April, 1951) describe experiments in which air was introduced intravenously into rabbits at controlled speeds and pressures. The pathological changes and certain functional disturbances in the cardio-vascular system due to this air embolism are discussed and contrasted with the effects of other types of embolism. Reasons are given for excluding vagal inhibition of coronary flow, obstruction of the coronary veins by air bubbles, coronary arterial embolism and primary cerebral ischæmia as factors in the circulatory upset, and it is concluded that death results from pulmonary block by air bubbles. Attention is drawn to the tolerance rabbits show for sudden ligation of both carotid arteries, probably because of an efficient vertebral circulation.

MORPHOLOGY.

Zona Glomerulosa of Bovine Adrenal.

A. F. WEBER *et alii* (*Journal of Morphology*, November, 1950) state that while earlier investigators working with various animal species have found that the cells of the adrenal cortex and in particular those of the *zona glomerulosa* show changes in number, size and cellular components during the œstrous cycle and pregnancy, little study has been made of the bovine adrenal cortex, although it is especially suitable for study of this kind. The present work was done to study the cellular structure and arrangement in the *zona glomerulosa* and to determine whether demonstrable histological changes occurred in this layer under various physiological conditions. It is believed that this study will aid in the understanding of the relationship of the adrenal to the physiology and pathology of reproduction. Studies were made of adrenal cortices in over 200 specimens from the four-month fœtus to adult cattle; these were male, bulls and steers, and female, both nulliparous as well as multiparous, pregnant and non-pregnant cows. The thickness of the *zona glomerulosa* was determined, and the occurrence and nature of certain cytoplasmic particulates were studied. In two five-month-old fœtuses the *zona glomerulosa* was set apart distinctly from the inner cortical zone, while that of the four-month-old fœtus tissue was apparently undergoing incorporation from the glomerulosa into the fasciculata. This latter change was prominent in the glands of the three-month-old fœtus. In three-weeks-old calves the *zona glomerulosa* was entirely separated from the fasciculata by a continuous connective tissue layer. The continuity of this connective tissue layer could not be demonstrated in older cattle. The incorporation of glomerulosa cells into the fasciculata was slight. Three sources of evidence for this view are suggested: (i) cytoplasmic granules were confined to the glomerulosa zone except for their sporadic appearance in nests of fasciculata cells adjacent to this zone; (ii) degeneration of glomerulosa tissue occurred exclusively of any signs of pyknosis and vacuolization of adjacent fasciculata tissue; (iii) when fat stains were employed the margin of the diffusely red-staining tissue concurred strictly with the boundaries of the fasciculata. Two types of cytoplasmic granules were found in the glomerulosa cells. The common one was spherical and retained orange G, but did not stain with Janus green B. The other was rod-shaped, retained acid fuchsin more strongly and stained with Janus green B. Both types stained black when subjected to the technique for phospholipids. In the adrenals of steers there was a greater number of cytoplasmic granules than in animals of the other groups. Soluble lipids were found in small quantity in the glomerulosa cells. No positive results from tests for cholesterol were obtained with any of the glands studied. Sloughing of large quantities of glomerulosa cells into the medullary venous sinuses was clearly demonstrated. Approximately one out of every three adrenal glands from adult cattle contained from one to several areas of extramedullary hæmatopoiesis. This process involved the entire cortex and was associated predominantly with granulocyte production.

Nerves of Atrio-Ventricular Bundle.

E. J. FIELD (*Journal of Anatomy*, April, 1951) describes the nervous component of the atrio-ventricular bundle in the sheep, rabbit, guinea-pig, rat, cat, dog, macaque, chimpanzee and gorilla and in man. The nerve fibres within the bundle are regarded for the most part as post-ganglionic autonomic fibres regulating conduction of the cardiac impulse carried by the specialized muscle of the atrio-ventricular bundle. The author states that the relation between the nervous and muscular components of the bundle is so close that it is impossible by experimental means to destroy one without the other. For this reason, it is difficult to ascertain the exact parts played by the muscle and nerve components; but examination of clinico-pathological material tends to support the view that they have an important role in cardiac action.

Synaptic End-Bulbs.

R. A. HAGGAR AND M. L. BARR (*Journal of Comparative Neurology*, August, 1950) have determined the size distribution of synaptic end-bulbs in the seventh lumbar and first sacral segments of the spinal cord of a cat. They state that there is considerable variation in size of endings within the same cell group and even on the same cell. In general, the end-bulbs are largest on cells of the dorsal horn, becoming progressively smaller on cells of the medial, ventro-lateral and dorso-lateral groups of the ventral horn. The data are offered as a standard of comparison for future work on the experimental pathology of the synapse and particularly for the valuable, but insufficiently exploited, method of end-bulb degeneration for neuroanatomical studies. A method of preparing scale models of nerve cells is described briefly, in the hope that such fairly accurate cell models may help to fill the need for three-dimensional illustration of the nerve cell and its synapses.

Subclavian Origin of Bronchial Arteries.

R. O'RAHILLY *et alii* (*The Anatomical Record*, October, 1950) describe the variability of the bronchial arteries and their embryological development, but state that the topography of these variations is usually omitted in descriptions in the literature. The authors present these details of a particular case and submit photographs and key diagrams.

Nutrition of the Ovum.

R. M. WOTTON AND P. A. VILLAGE (*The Anatomical Record*, June, 1951) have experimentally followed up a suggestion that nutrition of the egg cell during a major portion of its development is closely associated with the activity of certain accessory nurse cells. In a series of 40 kittens the authors studied the cellular channels through the *zona pellucida* by which the oocyte may receive food substances. These were found to be projections belonging to two types of accessory nurse cells situated in the follicle wall. By following the path of previously stained cod liver oil from the blood capillaries into the ovum the authors established the transport function of these accessory cells for this substance, and presumably for other materials needed by the ovum.

Anomalous Vertebræ from Human Lumbo-Sacral Column.

G. G. ROWE (*The Anatomical Record*, June, 1950) describes three specimens with the same unusual variation, namely, a loss of continuity between the superior articular process and the pedicle of the first sacral segment. In two of these sacra, *spina bifida* involved the same neural arch, and thus one-half of the arch was free of any bony connexion. One case is presented of unilateral rudimentary or absent right inferior articular process of the fifth lumbar vertebra. One case is described of separation between the lamina and mammillary process of the fifth lumbar vertebra. The probable embryological basis for these variations is discussed.

Ventral Horn Cells.

J. M. SPRAGUE (*Journal of Comparative Neurology*, August, 1951) presents evidence to show that cells of the ventral horn of the spinal cord cannot be classified functionally on the basis of their topographical position, or on their morphology. The larger propriospinal cells are morphologically indistinguishable from the motor cells and both of these in turn from the border cells of Cooper and Sherrington.

Special Articles for the Clinician.

(CONTRIBUTED BY REQUEST.)

VII.

THE SIGNIFICANCE OF MENORRHAGIA AND METRORRHAGIA.

DEFINITION.

By menorrhagia is meant excessive loss of blood at the menstrual period, that is to say, excessive for that particular individual. The loss may be of all degrees of severity, is often prolonged, and is sometimes associated with short or irregular cycles. On the other hand metrorrhagia means intermenstrual hæmorrhage from the uterus, but clinically the two conditions frequently coexist and may be due to the same cause but not necessarily so. However, if the definition of metrorrhagia is broadened to include all hæmorrhage from the uterus not directly concerned with the menstrual function, then the aberrations of conception such as abortion, ectopic pregnancy, hydatidiform mole and chorionepithelioma must be considered in such a review as this, as well as those conditions that cause uterine bleeding after the menses have finished or during an interval of amenorrhœa.

Perhaps it would be as well in an article of this kind to refer briefly to the transient bleeding occasionally noted in the newborn. This is not, of course, precocious menstruation, but an œstrogen withdrawal bleeding, contingent on the loss of maternal hormone from the fœtal circulation. Later, in childhood, uterine bleeding usually does denote the precocious onset of menstruation, but rarely it may be due to a functioning tumour of the ovary, and still more rarely to the *sarcoma botryoides* of the cervix and other types of uterine malignant disease. Later still, during reproductive life, the intermenstrual ovulation bleeding occurring at the middle of the menstrual cycle presumably results from the temporary fall in the effective œstrogen concentration obtaining at the time immediately following follicle rupture. The amount of bleeding varies from a microscopic amount to that sufficient to simulate a period and is usually accompanied by pain in one or other iliac fossa.

MENORRHAGIA.

A woman with menorrhagia usually seeks advice for two reasons, firstly because the perceptible change in the menstrual loss may disturb her, and secondly because of such symptoms as listlessness, tiredness or shortness of breath. However, it must be emphasized at the outset that menorrhagia is not a clinical or pathological entity, but always a symptom, and that before any treatment can be undertaken the underlying cause must be elucidated; and that furthermore sometimes there may be a combination of ætiological factors. To this end, the first step is to obtain a careful history and particularly an accurate menstrual history, in order to assess the severity of the condition and its type; and this in itself is by no means easy. Then follows a general physical examination, pelvic examination including careful inspection of the cervix in a good light, and such special investigations as are deemed necessary. This must be supplemented sooner or later by a careful, thorough diagnostic curettage, and in some instances by hysterography as well. By this time most cases will have resolved themselves broadly into those due to some local condition and those in which no gross pelvic pathological change is apparent. First let us consider the latter category.

Menorrhagia Associated with no Obvious Pelvic Lesion.

An acute febrile illness such as typhoid fever, pneumonia and particularly influenza may be responsible for menorrhagia, and the same can be said for conditions associated with long-standing fatigue and debility, and even chronic constipation. Chronic constitutional diseases such as cardiovascular disease, hypertension, diabetes, tuberculosis, hepatic cirrhosis and nephritis, occasionally give rise to menorrhagia, and the same applies to chronic intoxications such as alcoholism and lead poisoning. However, in conditions producing debility, amenorrhœa is just as common as menorrhagia, if not more common.

The patient may be found to be suffering from myxœdema, although hyperthyreoidism also may be responsible for excessive menstrual loss. However, in the later stages of both these conditions amenorrhœa is characteristic. Conditions such as splenic anæmia, aplastic anæmia, lymphatic leuchæmia, thrombocytopenic purpura, and other such blood dyscrasias will be eliminated by a complete hæmatological examination. There may be a subclinical avitaminosis or even a manifest deficiency such as scurvy. Vitamin *B* deficiency may produce menorrhagia by interfering with the capacity of the liver to inactivate œstrogens. In recent years also, an entirely psychogenic basis for even severe and long-continued menorrhagia has been recognized as responsible in some cases, and this could be engendered by anxiety states, marital unhappiness, any form of emotional stress, uncongenial environment *et cetera*.

In all of the above conditions the history and relevant special investigations will finally determine the correct diagnosis, but this may be very elusive.

The Menorrhagia of Puberty and the Menopause.

The inception of menstruation is often associated with severe floodings and irregular menstruation, usually anovular in type and painless. One or two years may elapse before normal flow is established. However, bleeding dyscrasias must be excluded because menorrhagia may be the sole manifestation of the hæmorrhagic tendency. A moderately excessive loss is exceedingly common, but occasionally the bleeding assumes serious proportions. With the efflux of time the natural tendency is towards restoration of normal ovulatory function and, therefore, spontaneous cure of the irregular cycles and menorrhagia. Similarly, at the other extreme of reproductive life, in the premenopausal and menopausal years, faltering ovarian activity associated with the involution of the endocrine system frequently leads to menorrhagia and irregular, usually anovular, menstruation. However, it must be remembered that intractable menopausal bleeding requiring intervention is seldom purely functional and a persistent flow points to a pathological condition.

Functional Uterine Bleeding.

Although functional uterine bleeding is defined as bleeding from the uterus in the absence of an obvious organic cause, it is generally understood to refer only to bleeding of an ovarian endocrinopathic nature. It occurs at any age during reproductive life, particularly the latter years, and it includes the menorrhagia of puberty and the menopause already discussed, and these together make up about 60% of the cases. The bleeding, of all degrees of severity and usually painless, may be menorrhagic or metrorrhagic, or a combination of the two, although the periodic type is perhaps more characteristic. The menstrual rhythm may be maintained as usual or speeded up, or retarded in such a way that the floodings may be separated by non-bleeding phases of varying and considerable duration. This last-mentioned feature is particularly applicable to the menopausal and puberal types. Schröder's *metropathia hæmorrhagica*, in which failure of follicle rupture and absence of functioning *corpus luteum* lead to abnormally persistent and excessive œstrogenic stimulation of the endometrium, is still the accepted mechanism for perhaps the largest number of cases, the bleeding phases corresponding with periodic drops in the relative œstrogen levels. However, not all cases of functional uterine bleeding can be fitted into the one mould and there are many discrepancies and gaps in our knowledge. As if to prove that the endocrine disturbance is not identical in all cases, the bleeding may occur from any type of endometrium. Even post-menopausal bleeding is occasionally functional, but the vast majority of cases have an organic basis and should be considered as such until proven otherwise, and even so, endometrial hyperplasia, even though in itself benign, is regarded by many as potentially dangerous from the point of view of malignant disease. Needless to say, and irrespective of the age of the patient, the diagnosis of functional uterine bleeding remains merely presumed until its essentially functional nature has been established by careful, thorough curettage of the uterine cavity and histological examination of the endometrium. In this way, and only in this way, unsuspected organic conditions of the uterine cavity can be discovered, and information obtained about a benign endometrium without which any endocrine therapy that may be contemplated cannot be rationally applied. Biopsy may reveal endometrial hyperplasia or atrophy, irregular ripening or irregular shedding of the endometrium, or it may be normal. Irregular shedding is characterized by a prolongation of cyclic menstrual bleeding from progestational endometrium, with often in addition an increase in the amount of the bleeding. Although the big majority of cases of functional bleeding are associated with anovular cycles, little is known of the cause of the excessive menstrual flow sometimes occurring with ovular cycles.

In addition to the help given by the menstrual history and endometrial biopsy, basal temperature records may give further information as to the type of bleeding pattern one is dealing with, the biphasic chart indicating that the cycle is ovulatory.

Having discussed bleeding due to general causes and endocrinopathies, let us now turn to a consideration of what local lesions might be responsible for menorrhagia.

Menorrhagia due to Local Pelvic Conditions.

Broadly speaking, any condition causing increase in the area or thickness of the endometrium, or interference with the normal contractility of the uterine muscle, will cause menorrhagia. If the condition has followed an abortion or confinement, particularly if complicated by infection, the cause probably lies in retained gestational products, and the uterus will be found to be enlarged, subinvoluted, and probably but not necessarily retroverted.

Progressively increasing menorrhagia always suggests pelvic pathological change, and the two conditions most commonly responsible are uterine fibroids and endometriosis. Submucous fibroids, particularly if pedunculated, bleed because of passive congestion, necrosis and ulceration. Large submucous and interstitial fibroids cause increase in the size of the uterine cavity and, therefore, of the bleeding surface. Subperitoneal tumours do not tend to affect menstruation, and indeed, when abnormal bleeding occurs with such tumours, one should suspect and look for the presence of some other lesion to account for it. In any case it should always be borne in mind that the fibroids may be incidental to the bleeding, particularly if there is intermenstrual hæmorrhage as well, or if the bleeding occurs after the menopause. Furthermore, associated ovarian dysfunction may be a contributing factor in these cases. Metrorrhagia due to fibroids usually suggests that one or more of the tumours is becoming submucous. Post-menopausal increase in size of a fibroid is very suspicious of malignant degeneration, particularly if associated with bleeding.

If endometriosis (including adenomyosis) is responsible, the patient, usually a woman over the age of thirty years, may complain of acquired and progressive premenstrual dysmenorrhœa, deep dyspareunia, pain in the lower part of the abdomen, sacral and coccygeal areas, and often "jabbing" rectal pain mainly with defæcation and at the menses.

Chronic pelvic inflammation, of any variety and from any cause, may closely simulate endometriosis in symptomatology, and is almost always associated with menorrhagia. Genital tuberculosis, comparatively rare in Australia, is usually associated with menorrhagia, but may sometimes (up to 30%) be responsible for amenorrhœa or oligomenorrhœa. The primary focus, usually tubal, is itself secondary to tuberculosis elsewhere, for example, lung or kidney. Chronic metritis, *fibrosis uteri* and chronic subinvolution form a group of conditions responsible for menorrhagia, but their ætiology is still very much *sub judice*. However, it is generally thought that most of these cases of chronic subinvolution are the result of an endocrine influence causing changes similar to *metropathia hæmorrhagica* and that true chronic metritis of infective origin is rare. The uterus is bulky and of variable consistency and may be retroverted.

If the menorrhagia is associated with a blood-stained intermenstrual discharge one's suspicions of malignant disease of the cervix or *corpus uteri* should be aroused and, irrespective of the patient's age, must be excluded before proceeding further, because it must be remembered that malignant disease of the uterus can occur considerably before the age of forty and also in the absence of palpable pelvic disease.

One is not impressed that retroversion of the uterus *per se* has any effect on menstruation, but it may be associated with infection and subinvolution. Uncomplicated non-functioning tumours of the ovary have no significant effect.

METRORRHAGIA.

Unlike menorrhagia, in which the causes are frequently of general origin, those responsible for metrorrhagia are usually to be found in the pelvis. However, the two conditions, as already stated, may coexist and in these circumstances may or may not have the same pathological basis. In every instance the onus is on the practitioner to exclude malignant disease. Submucous fibroids, fibroid polypi, mucous and endometrial polypi, retained products of conception and carcinoma are the commoner causes emanating from the uterine body. The uterine polypi may protrude into the cervix or vaginal canal, in which case bleeding, due to necrosis and ulceration, is a common symptom. The amount of bleeding from retained products bears no relationship to the amount of tissue still in the uterus. Endometrial tuberculosis, discussed elsewhere, is a rare cause. If the bleeding comes from the cervix, examination may reveal an erosion or a polypus, decubitus ulceration accompanying procidentia, or an ulceration due to foreign bodies, pessaries *et cetera*. Or it may be due to malignant disease. Inflammation, torsion or other complication of the adnexæ may cause uterine bleeding and disturbance of menstrual rhythm, due more to ovarian dysfunction than to other reasons. Apart from those which produce œstrogen, ovarian tumours do not as a rule give rise to external bleeding. However, the extremely rare carcinoma of the Fallopian tube is not infrequently associated with bleeding. Acute vaginitis (for example, trichomonas or pyogenic infection) may cause blood-staining of a discharge. Œstrogen treatment of the menopausal syndrome may in itself be responsible for intermittent uterine bleeding due to fluctuations in the blood œstrogen level, but again this cannot be accepted as a diagnosis until malignant disease has been excluded. In the reproductive age, amenorrhœa of a few to several weeks, followed by slight uterine bleeding and associated with unilateral pelvic pain and adnexal mass, should always lead to the suspicion of tubal gestation. The bleeding of hydatidiform mole appears in the early months of pregnancy and is accompanied by a disproportionately large uterus and high titres of chorionic gonadotropin. Chorionepithelioma manifests itself by a persistence of bleeding after the evacuation of a hydatidiform mole, or after miscarriage or full-term delivery, and also typically gives characteristic results to quantitative pregnancy tests. Both of these two last-mentioned conditions are rare, however, particularly chorionepithelioma.

By far the most important cause of uterine bleeding, from the standpoint of its life-and-death significance to the patient, is that due to uterine malignant disease, particularly cancer of the cervix. Corpus carcinoma develops mainly after the menopause, causing at first only a slight and occasional blood-tinged watery discharge, later becoming more persistent and profuse. On the other hand cancer of the cervix affects the younger age groups and is much more common, so much so that its possible presence must be constantly kept in mind. Typically there will be intermenstrual "spotting" or contact bleeding. As stated elsewhere in this article, no pelvic examination is complete without the careful examination of the cervix in a good light, and no apology is made for reiterating this elementary rule of procedure. Any suspicious condition will call for a biopsy, but the discovery of a benign lesion of the cervix, such as, for example, a polypus, which could cause the bleeding, does not obviate the necessity for exploration of the uterine cavity, as well, by means of curettage.

The elucidation of the cause of menorrhagia or metrorrhagia may be a simple matter, or on the other hand extremely difficult. It would appear almost without exception that careful and thorough curettage and histological examination of the endometrium are, among other things, essential before the significance of the bleeding can be properly assessed. Whether the mass periodic routine pelvic examination of healthy symptomless women is practicable, or even desirable, in an effort to detect early cancer or precancerous lesions of the genital tract, has been a subject for some controversy. Be this as it may, the inescapable fact remains that, when patients do eventually present themselves with conditions such as menorrhagia or metrorrhagia, while it behoves us, of course, to determine the cause as soon as possible, the early exclusion of malignant disease must remain our primary responsibility and we must be always cognizant of this.

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British Medical Association News.

SCIENTIFIC.

A MEETING of the Victorian Branch of the British Medical Association was held at Prince Henry's Hospital, Melbourne, on September 19, 1951. The meeting took the form of a series of clinical demonstrations by members of the medical and surgical staff of the hospital.

Suspected Myelosclerosis.

DR. THEO FRANK presented a single woman patient, aged fifty-nine years, a school teacher, who had reported on February 19, 1951, with a history of having had in December,

1950, treatment for cellulitis of both shin regions with a prompt response to penicillin injections and rest. On February 16 a similar reddened area had recurred on both legs, much worse on the right. Apart from chilblains on the back of both legs in winter, she had enjoyed good health. Her father had died from angina at the age of seventy-five years, whilst her mother had died of leuchæmia at the age of eighty-three years. Three sisters and one brother were alive and well. The only abnormal findings on clinical examination were pallor, gross piles, a firm spleen palpable four fingers' breadth below the left costal margin, thickened skin of the lower third of both legs and a swelling around the left ankle. No glands were palpable. From that examination a provisional diagnosis of chronic myeloid leuchæmia with leucemides was made. Examination of the blood on February 21 revealed a hæmoglobin value of 70% (9·9 grammes per 100 millilitres of blood), a total erythrocyte count of 3,300,000 per cubic millimetre and a total leucocyte count of 5700 per cubic millimetre. The colour index was 1·03%. A differential count of the white cells showed 47% polymorphonuclear cells, 15% of old metamyelocytes, 4·5% of young metamyelocytes, 15·5% of lymphocytes, 8% of eosinophile cells, 3% of basophile cells and 7% of myelocytes. In the blood films the red cells were well hæmoglobinized, but showed a fair amount of anisocytosis, the size varying from moderate macrocytes to pronounced microcytes, although many normocytes were present. Three erythroblasts were seen during the counting of 200 nucleated cells. Reticulocytes were less than 1% of the total red cells. X-ray examination after a barium meal on March 16 showed no lesion in the gastro-duodenal area; in addition an excretion pyelogram was also normal. In the X-ray films the mass on the left side of the abdomen was typical of an enlarged spleen. Bone marrow examination on March 5 showed a slight increase in neutrophile cells only, but no evidence of leuchæmia. The following differential count of bone marrow cells was obtained: myeloblasts 2·0%, premyelocytes 1·5%, myelocytes 5·0%, young metamyelocytes 7·0%, band metamyelocytes 16·0%, neutrophile cells 24%, eosinophile cells 3·5%, lymphocytes 14%, monocytes 1·5%, megaloblasts 0·5%, proerythroblasts 1·5%, erythroblasts 10%, normoblasts 11%, reticular cells 0·5%, megakaryocytes 0·5%, hæmatocytoblasts 1·0% and Türk cells 0·5%. A further blood count on April 26 yielded a similar result to the previous one. The last available count made on August 2, 1951, revealed a hæmoglobin value of 59% (8·3 grammes per 100 millilitres), a total erythrocyte count of 3,050,000 per cubic millimetre, a total leucocyte count of 7600 per cubic millimetre and a colour index of 0·98%. Examination of the red cells showed a wide degree of anisocytosis, some poikilocytosis and a mild degree of anisochromia, the general tendency being towards slight hypochromia. Reticulocytes numbered approximately 2·5%. Three normoblasts and one late erythroblast were seen during the counting of 200 leucocytes. The platelets were moderately plentiful but somewhat irregular in shape and size. The differential leucocyte count showed a slightly increased shift to the left in the granular series. The following conclusions were recorded by the hæmatologist: "A peripheral leuco-erythroblastic anæmia of this type with a normal bone marrow picture, together with splenomegaly and relatively mild clinical symptoms is compatible with a diagnosis of myelosclerosis. A chronic hæmolytic process, which can occur without obvious jaundice, is also a possibility, but less likely than myelosclerosis." The blood fragility of the red cells was increased with hæmolysis beginning between 0·56% and 0·60% dilutions of saline (the upper level of normal being about 0·50%). The Coombs test result for hæmolysins was negative. The test for urinary urobilin showed a very small excess, whilst there were 30 milligrammes of stercobilin in 100 millilitres of fæces. An X-ray examination of the long bones showed a little patchy relative rarefaction in the upper end of the right humerus, with a similar, though less pronounced, appearance on the left side; the finding was probably of no clinical significance. Dr. Frank said that from the hæmatological findings, the most likely diagnosis appeared to be myelosclerosis, although an atypical hæmolytic anæmia could not be excluded. A further examination of a smear and section of the bone marrow would be helpful. In myelosclerosis there was an increased density in the bones due to an excess production of fibrous (myelofibrosis) or bone tissue (osteosclerosis); as a result, the normal blood-forming tissue in the marrow was replaced by fibrous tissue. The diagnosis depended at times on examination of a histological section, which demonstrated much fibrosis and numerous megakaryocytes. The disease often ran a long course, and the only form of treatment which was effective was blood transfusion for the severe anæmia. Splenectomy was contraindicated and often fatal, as the splenic enlargement was compensatory and usually an important site of hæmopoiesis.

Polycythæmia Vera Treated with Radioactive Phosphorus.

Dr. Frank's second patient, a man, aged sixty-five years, formerly a storeman and packer, had been admitted to Prince Henry's Hospital on May 15, 1951, and had stated that apart from a stroke nineteen years previously, which had affected the right side of his body and lasted for three days, he had enjoyed excellent health. In February, 1950, he had suffered a right hemiplegia, a sequel of cerebral thrombosis; that had gradually decreased, although there was still weakness in the right arm and right leg. As long as he could remember, his complexion had been high coloured. About five months before he had noted distension after meals, epigastric discomfort unrelated to food and nausea on smelling food; all those symptoms promptly subsided with antacid powder. For the past twelve months he had been short of breath, although he could still do heavy lifting. About five months before there had been a spontaneous extensive hæmorrhage in the right leg, which caused severe bruising. Examination of the patient showed a highly coloured dusky appearance of the face and hands, arteriosclerosis with a systolic pressure of 210 millimetres of mercury and a diastolic pressure of 110 millimetres, retinal arteriosclerosis and a right hemiparesis. Both the spleen and liver could be palpated two fingers' breadth below the costal margins. A blood examination on May 16 showed a hæmoglobin level of 160% (23 grammes per 100 millilitres) and total counts of 8,700,000 erythrocytes and 14,300 leucocytes per cubic millimetre. The mean corpuscular volume was 94·2 cubic μ, the mean corpuscular hæmoglobin concentration 29·2% and the hæmatocrit reading 82%. A differential count of the leucocytes was within normal limits. A blood smear examined was normal, apart from one nucleated red cell in a count of 100 nucleated cells. There were 287,000 platelets per cubic millimetre. The bleeding time (Duke's method) and the coagulation time (Lee and White venous method) were two minutes thirty seconds and six minutes respectively. Examination of a smear from the bone marrow revealed a normoblastic type of marrow with a considerable increase in the red cell and to a less extent in the white cell precursors. An X-ray examination of the chest revealed rounding of the left cardiac border consistent with ventricular enlargement and also venous congestion of the lung fields. The X-ray appearance after a barium meal was normal except for spasticity of the pyloric antrum, whilst the cholecystogram showed a sluggishly reacting gall-bladder. The fasting blood urea level was 65 milligrammes per 100 millilitres, whilst the urea clearance was 32% of average normal clearance. Moderate amounts of urobilin were present in the urine. Treatment at the start consisted of venesection of 16 ounces of blood on June 8, of 23 ounces on June 11 and of 30 ounces on June 14. A blood count on June 15 showed a hæmoglobin value of 138% (19·5 grammes) per 100 millilitres and total counts of erythrocytes 6,800,000, leucocytes 7800 and platelets 546,000 per cubic millimetre. On June 28 4·4 millicuries of radioactive phosphorus (P^{32}) in nine millilitres of water were given orally on an empty stomach. On July 31 the patient reported that his blood was thinner after he cut his finger and that his health was improved. When he was examined on September 5, improvement in health had occurred, the spleen was impalpable, whilst the liver was palpable one finger's breadth below the left costal margin. A blood examination on that date showed a hæmoglobin value of 120% (17·5 grammes *per centum*) and total counts of erythrocytes 6,360,000, leucocytes 5000 and platelets 268,000 per cubic millimetre.

Dr. Frank said that in the treatment of the disease, the first essential was to be certain of the diagnosis and to exclude polycythæmia secondary to other diseases, such as congenital heart disease and emphysema, and also the type associated with hypertension, namely, *polycythæmia hypertonica* of Gaisbock. Many different types of treatment had been tried—namely, repeated venesections at different intervals, administration of phenylhydrazine or acetylhydrazine, administration of Fowler's solution and irradiation of long bones and other bones concerned with erythropoiesis, and all had produced, with adequate control, good results. At the present time the consensus of opinion was that radioactive phosphorus probably gave the most satisfactory results. Lawrence, who had treated a series of 121 patients, had noted the following facts: (i) Palpable spleens which were present in 65% of cases became smaller or disappeared after therapy. (ii) There was a lessening of immature cells in the blood picture. (iii) A definite fall in

blood pressure occurred in one-third of the cases. (iv) Remissions of from three to eight years occurred. (v) Thrombosis occurred in only 4% of cases, whereas with other forms of treatment it had been as high as 25%. (vi) The incidence of leuchæmia development had not increased. Some authorities had suggested that a combination of treatment namely, venesection at the start, followed by administration of radioactive phosphorus, gave the best results. In the hands of certain investigators, nitrogen mustard given intravenously had also produced good results, but larger series of cases were essential before one could be sure of its ultimate value.

Toxic Thrombocytopenic Purpura, Diabetes Mellitus, Chronic Infective Arthritis, Bacilluria.

Dr. Frank then presented a married woman, aged thirty-four years, who when first examined by him on November 24, 1949, had a history of exhaustion, stiffness of the hands, swelling of the proximal interphalangeal joints and pains in the lumbar part of the spine of four months' duration. For the past sixteen years she had suffered from *diabetes mellitus*, which was fairly well controlled by diet and insulin. Seven years before she had been in hospital for two months with pyelitis. The family history was clear of abnormality. Routine examination of the patient revealed chronic tonsillitis, endocervicitis, pronounced bacilluria, a blood pressure of 160 millimetres of mercury, systolic, and 100 millimetres, diastolic, and typical changes of chronic infective arthritis in some of the proximal interphalangeal joints of the hands. An excretion pyelogram revealed blunting of the renal calyces and arteriosclerosis of the pelvic vessels. In view of the joint changes the tonsils were removed and the *cervix uteri* was cauterized on January 25, 1950. During the next months there was a gradual improvement in the joints and the patient felt much better. However, in October, 1950, the feet became increasingly painful, and relief was obtained with suitable physiotherapy. In January, 1951, the interphalangeal joints of the hands again became painful and swollen, and as a result a course of gold injections (0·1 gramme) was given weekly from March 2, 1951, for six injections. Treatment was then stopped (on April 12) because of itchiness of the skin and a trace of albumin in the urine; there had been a definite lessening of the rheumatic pains. On May 17 (five weeks later) the patient was examined again. She had a history of lumbar backache and of pains in the right iliac fossa for six days, of hæmaturia for five days and of nocturnal frequency of micturition for fourteen days. The urine on microscopic examination was found to be loaded with pus cells, red blood cells and bacilli, and a diagnosis of bacilluria was made. Treatment with alkalis and sulphadiazine (eight tablets daily for six days) rapidly cleared up the urinary infection. However, two weeks later (seven weeks after cessation of the gold injections) multiple purpuric spots on the chest wall, tongue and left conjunctiva, ecchymoses at the site of insulin injections and petechiæ on lips and arms developed. As a result the patient was admitted to Prince Henry's Hospital on June 9, 1951, for observation. A blood examination on June 14 showed a *hæmoglobin value of 82% (11·5 grammes per centum)* and total counts of 4,030,000 erythrocytes, 6800 leucocytes and 24,000 platelets per cubic millimetre. The bleeding time by Duke's method was six minutes thirty seconds, whilst the coagulation time (Lee and White venous method) was seventeen minutes. Clot retraction was minimal after eighteen hours. Culture of the urine yielded a profuse growth of *Bacillus coli communis*. The blood fragility test result was normal, whilst a bone marrow biopsy on June 29 showed the marrow to be within normal limits apart from the finding that few megakaryocytes showed evidence of platelet production. Whilst under observation, the patient suffered from epistaxis and menorrhagia. Numerous platelet counts showed a level ranging from 24,000 to 34,400 per cubic millimetre, and on her discharge from hospital on August 2 her platelet count was 26,390 per cubic millimetre. However, a month later (August 30) there were 158,000 platelets per cubic millimetre.

Dr. Frank said that the patient suffered from toxic thrombocytopenic purpura apparently due to intoxication from gold therapy for her chronic infective arthritis. It had been shown that the administration of BAL (British anti-Lewisite) had produced good results in cases of dermatitis, thrombocytopenic purpura and granulocytopenia due to intoxication from gold therapy, for the rate of excretion of gold in the urine was increased. BAL should be given early in the toxic reaction if it was to be effective. In severe reactions, it had been suggested that three milligrammes of BAL per kilogram of body weight should be given every four hours for twelve injections; four injections were given on the third day, and then two injections for ten days or until complete recovery. Gold therapy must be immediately discontinued in the presence of toxic reactions.

Hæmolytic Anæmia.

Dr. Frank's next patient, a male clerk, aged thirty-six years, had been examined by him on July 27, 1951. The patient had then stated that an enlarged spleen had been noted at the age of eighteen years whilst he was suffering from pneumonia. A diagnosis of acholuric jaundice had been made by a consultant, and arsenic drops had been ordered. Apart from three attacks of pain over the splenic region during bouts of influenza and of streptococcal throat, his health had been good, with the result that, over a period of eighteen years, he had been away from work for only four weeks. About eight months before coming to Dr. Frank the patient had fallen onto the "end of his spine", and, as a result, his back had become stiff; the X-ray appearance of his spine was normal. However, as he looked pale, further investigation was ordered. There had been no history of sore tongue, of hæmorrhage or of hæmolytic crises. Apart from measles, mumps and streptococcal tonsillitis in 1946, his past history had been free of other abnormalities. Both his parents were alive and well. He had two sisters and one brother. His eldest sister, now aged thirty-three years, had been found to have an enlarged spleen at the age of twenty years and recently had needed blood transfusions for anæmia, the cause of which was indefinite. A careful investigation of his immediate relatives had failed to reveal any with splenomegaly. Apart from pallor and a spleen palpable four fingers' breadth below the left costal margin, routine medical examination of the patient failed to reveal any abnormality. A blood count carried out on May 30, 1951, showed a hæmoglobin value of 72% (10 grammes per centum) and total counts of 4,290,000 erythrocytes and 15,200 leucocytes per cubic millimetre. In the differential count, there were polymorphonuclear cells 58%, old metamyelocytes 3%, lymphocytes 37%, eosinophile cells 1% and monocytes 1%. Examination of a blood film showed that the red cells were fairly normal and did not have the usual morphological features of the microspherocytes seen in cases of acholuric jaundice. Stippled cells and 10% of reticulocytes were present, features indicative of increased erythropoietic activity as might be caused by a hæmolytic anæmia. The white cells were normal, although the total count was increased. Platelets were present in the films. A serum bilirubin estimation made on June 27, 1951, showed 3·0 milligrammes per 100 millilitres, whilst a Coombs test for circulating hæmolysins yielded a negative result. A blood fragility test on July 11 yielded a normal result: hæmolysis of the blood of both the patient and the control commenced at a dilution of 0·45% of saline and was complete at a dilution of 0·30%. A Wassermann test of the blood serum yielded a negative result. Examination of the bone marrow on August 7 revealed pronounced cellularity and an increase in erythropoiesis, findings consistent with a diagnosis of hæmolytic anæmia. In the bone marrow smear there were myeloblasts 1·5%, myelocytes 9·5%, young metamyelocytes 6·5%, band metamyelocytes 8·0%, neutrophile cells 15·5%, lymphocytes 1·5%, megaloblasts 0·5%, proerythroblasts 4·5%, erythroblasts 22·0%, normoblasts 31·0%, reticulum cells 1·0% and megakaryocytes 0·5%.

Dr. Frank said that the investigations had revealed a hæmolytic anæmia, the cause of which was unknown. There was no positive proof for the diagnosis of acholuric familial jaundice, for the blood fragility test result was normal, whilst the causation of splenomegaly in the patient's sister was still unproven. Splenectomy had been recommended when the disease was first recognized in the patient at the age of eighteen years; as his health had been good, the patient had refused surgery and took iron orally when he felt that it was needed. Should a hæmolytic crisis arise at any time, repeated transfusions of blood would be essential and the question of splenectomy would again arise.

Multiple Myelomatosis.

Dr. Frank's last patient, a grocer, aged sixty years, had been examined on June 22, 1951. His history was that five months previously he had developed an acute backache after feeling something "go" in his back whilst pushing a car. That had been followed by recurrent attacks of pain across the lumbar region and the hip joint. On April 10, 1951, an X-ray examination of his lumbar region had revealed spondylitis and sacro-iliac arthritis. Fourteen days before the time of examination a sudden dimness of vision had occurred in his right eye and examination had revealed thrombosis of a right retinal vein and retinal arteriosclerosis. In addition, pains in the shoulders and paræs-

thesia in both legs had developed. During the last twelve months his weight had increased by two stone, whilst his exercise tolerance had gradually lessened. Routine examination revealed pronounced pallor, unhealthy tonsils, bacilluria and tenderness to percussion over the lower thoracic and lumbar spinous processes. There was also restricted movement in the lumbar region of the spine. A blood examination on June 25, 1951, showed a hæmoglobin value of 44% (6·2 grammes per 100 millilitres), total erythrocyte and leucocyte counts of 2,500,000 and 5700 per cubic millimetre respectively, and a colour index of 0·88%. In the film, the red cells had slight anisocytosis, but were normal in shape and well hæmoglobinized, whilst the reticulocytes were within normal limits and platelets were plentiful. A differential count of the white cells showed polymorphonuclear cells 32%, old metamyelocytes 3%, lymphocytes 58%, eosinophile cells 3% and monocytes 4%. As no obvious cause was discovered for the anæmia, further investigations were carried out; X-ray examination of the lungs and of the alimentary tract after a barium meal yielded normal findings. In view of the persistence of backache, another X-ray examination of the lumbo-sacral region was carried out on July 26, 1951, and the following report was made: "Marked general osteoporosis with scattered areas of bone destruction throughout the lower ribs, lumbar spine and pelvis. In addition, there is a partial collapse of several vertebral bodies, especially thoracic 12 and lumbar 1. These appearances could be due to either widespread bony metastases or possibly myelomatosis." A blood Wassermann test gave a negative reaction.

In view of the features mentioned and of a syncopal attack, he was admitted to Prince Henry's Hospital on August 1 for further investigations. Apart from the previous radiological findings, X-ray examination of the skull, scapulæ, humeri and clavicles revealed multiple osteolytic areas up to one centimetre in diameter, appearances consistent with a diagnosis of multiple myelomatosis. The serum protein values were 11·7 grammes *per centum*, the albumin proportion being 2·6 grammes *per centum*, the globulin proportion 9·1 grammes *per centum*, and the albumin-globulin ratio 0·29. The fermel gel test result was positive in ten minutes, and the serum calcium level was 11·8 milligrammes *per centum*. Bence-Jones proteose was also demonstrated in the urine. Examination of a blood film on August 20, 1951, showed the presence of nucleated red cells, non-segmented polymorphonuclear cells and myelocytes, findings which were noted in leuco-erythroblastic anæmia. The bleeding time (Duke's method) was four minutes, whilst the clotting time (Lee and White) was six minutes. Clot retraction was normal. A bone marrow biopsy showed a cellular marrow with some increase in the erythroid element, but the main feature was the presence of a large number of cells resembling plasmocytes except for the clumped appearance of the nucleus. Those cells were probably plasma cells and were evidence in favour of the diagnosis of myelomatosis. An X-ray examination of the lungs on August 8, 1951, showed a right basal atelectasis, whilst a bronchoscopic examination showed no intrinsic lesion but a fixed carina with a widened angle, apparently due to pressure from enlarged mediastinal glands. The Mantoux test result was positive to a strength of 1:1000 old tuberculin. Examination of sputum revealed no acid-fast cells, no carcinoma cells and no pathogenic organisms. The basal sedimentation rate was 47 millimetres in one hour. An excretion pyelogram revealed a mild bilateral pelvic hydronephrosis, whilst microscopic examination of the urine showed some pus cells and epithelial cells, but no tubercle bacilli or pathogenic organisms were found.

Dr. Frank said that the radiological changes in the bones, the bone marrow biopsy and the hyperglobulinæmia were features that established the diagnosis of multiple myelomatosis. Treatment had consisted of symptomatic measures for relief of backache by rest in bed, by the administration of analgesics and by the ordering of a Taylor brace for the patient to wear in the erect attitude. The anæmia had been lessened by two series of blood transfusions of three pints, but the rise in hæmoglobin level had not been as high as was expected. No specific treatment existed, but stilbamidine and pentamidine had been used by various observers apparently with relief of pain in some cases. Urethane had also been given orally for eight to ten weeks, and according to J. Philip Loge and R. Wayne Rundles ("The 1949 Year Book of Drug Therapy", page 385), some patients had obtained great relief of pain and of fever whilst the blood picture improved, and there was a decrease in the abnormal plasma or myeloma cells in the bone marrow with no progression of the destructive lesion in serial X-ray films of the skeleton. The patient presented had been given four grammes of urethane daily (two half-gramme capsules four-hourly), and as happened frequently, nausea and abdominal discomfort had occurred. At the time of the meeting he was still under treatment with urethane, and arrangements were being made for a course of deep X-ray therapy for relief of pain caused by bony destruction.

The Treatment of Inguinal Hernia.

DR. DOUGLAS DONALD discussed the treatment of inguinal hernia. He first demonstrated the anatomy of the inguinal canal, the demonstration being the result of personal dissection of many subjects in which the findings were at considerable variance with popular teaching. On this basis a treatment of inguinal hernia was displayed. Also on this basis was shown the reason for recurrences after standard accepted operations.

Urological Cases.

DR. J. B. SOMERSET discussed four cases in which the operation of total cystectomy had been performed for carcinoma of the bladder. These had been selected at random from a series extending back over the past three years. The operation had, in all cases, been performed in two stages, the first stage consisting of bilateral ureterosigmoidostomy carried out through a mid-line suprapubic incision followed by removal of the bladder after three weeks or so through a transverse transrectus incision about two fingers' breath above the pubis.

Dr. Somerset said that the cases illustrated several points: Firstly, the common development of chronic thirst following this operation in spite of the absence of any evidence of infection of the kidneys or urænia; secondly, the comparative safety of the end-to-side anastomosis; thirdly, the advantage of the long transverse incision for removal of the bladder. In one case the patient had developed a secondary malignant nodule in the scar, which had been successfully removed; this implant had occurred despite the fact that the bladder had never been opened. Another case illustrated the dramatic retrogression in the size of the tumour which could follow diversion of the urinary stream.

All cases illustrated the comparative comfort of the patient and the good rectal control which were present after operation.

Rheumatic Fever Treated with Cortisone and ACTH.

DR. T. DUDLEY HAGGER presented patients who had had rheumatic fever treated with cortisone and ACTH. He pointed out that the true place of these hormones in the prevention of heart damage in rheumatic fever could not be known for a number of years. Important claims had, however, been made from North America, and he thought that some members would be interested in seeing the immediate effects of the substances on rheumatic patients. It was apparent that their use could suppress fever, pain and joint swelling, perhaps a little more rapidly and completely than salicylates. The first patient, a girl, had been aged seven years when admitted to hospital on August 13, 1951, in her first attack of acute rheumatic fever. For five days she had had pain and swelling in various joints, and she had a temperature of 103° F. She was given no salicylates, but commenced ACTH therapy on the day of admission. Within twenty-four hours her temperature was normal, and several joints which had been swollen and very painful on admission had returned to a clinically normal condition. At the end of five weeks' treatment her erythrocyte sedimentation rate had fallen from 56 millimetres in one hour (Wintrobe) to 11 millimetres, and her cardiac signs had improved. It could not be claimed that the activity of the rheumatic process had ceased, or even that she was any better off than she would have been on salicylate therapy.

Another patient, however, was a boy, aged eleven years, who had been admitted to hospital on April 23, 1951, in his second attack of rheumatic fever. After six weeks of salicylate therapy in standard dosage his heart had increased in size, his systolic murmur was louder than on his admission to hospital and his erythrocyte sedimentation rate had remained over 50 millimetres in one hour. The salicylates were then stopped and he was given cortisone. Within a fortnight his sedimentation rate came down to eight millimetres in one hour, and at the time of the meeting, three weeks after cessation of cortisone treatment, it was 22 millimetres in one hour; heart size and pulse rate were normal, though the murmur had not decreased. In his case it appeared that cortisone had been more effective than standard doses of salicylates in combating the rheumatic process, and there seemed some reason to hope that his heart might have been shielded from some of the adverse effects

of the process. But until such time as it had been possible to follow over a period of a number of years carefully controlled groups of patients with and without hormone treatment, one could not really know whether the new drugs offered a real advance in methods of treating rheumatic fever.

Sarcoidosis.

DR. BRUCE ROBINSON showed patients to illustrate the features of sarcoidosis. The first was a married woman, aged fifty-eight years, who had had evidence of sarcoidosis for twenty years with involvement of skin, lymph nodes, eyes, lungs and bones. At present the condition appeared to be clearing, for although the X-ray film of the lungs still showed diffuse mottling, that of the phalanges showed evidence of recalcification. The eye lesions were inactive and those of the skin much less disfiguring. The patient had been given calciferol, but it was stopped after nausea developed.

The next patient was a single woman, aged forty-two years. At present the only evidence of sarcoidosis was in the X-ray film of the chest and in a fading skin lesion on the left temple. However, in the past she had had generalized symptoms with loss of weight, enlarged lymph nodes, iritis, painless swelling of the parotid glands and bilateral facial palsy. She had been taking calciferol, 50,000 international units daily, during the period of improvement.

The third patient, a married woman, aged twenty-nine years, was shown to illustrate the degree of apparent good health with absence of signs and symptoms possible in sarcoidosis. One year previously her condition had been suspected following a routine X-ray examination. At that time enlarged lymph nodes and a few skin sarcoids were found. At present some resolution appeared to have occurred in the lungs, and no other evidence of any disease was to be found.

Dr. Robinson's fourth patient, a man, aged forty-eight years, showed the gross damage that might be caused in the eyes. Following bilateral irido-cyclitis with bilateral facial palsy eight years before, he had gross corneal scarring, for which a partial tarsorrhaphy had been performed. In his case the Mantoux test result was positive, in the other it was negative.

The last patient was a man, aged sixty-three years, who for six years had had swollen ankles. At first they were regarded as tuberculous, but no cause could be found for this. A biopsy of the thickened periosteum showed non-specific chronic inflammatory changes. Four years before the time of the meeting he had developed skin sarcoids, which were still present despite calciferol therapy.

Dr. Robinson pointed out that the patients shown exemplified the protean manifestations of sarcoidosis and that the common syndromes were illustrated. Each patient showed evidence of a generalized disease of a chronic nature, causing little inconvenience except in the case of the patient with the damaged eye. All had been investigated, as far as was possible, to exclude other causes for the lesions. A confirmatory biopsy had been obtained from each. Dr. Robinson said that of the various treatments tried, calciferol appeared the most effective. He had not used cortisone and felt that it might be dangerous to do so. Streptomycin had had no apparent effect.

Chronic Miliary Tuberculosis.

DR. DOUGLAS GOULD presented a married woman, aged thirty years, who had been first admitted to hospital in June, 1948, when she was seven months pregnant. She complained of pleuritic pain on both sides for two weeks. She was regarded as having pneumonia, but there was little response to chemotherapy. The baby was born in July, one month prematurely, but the pains in the chest continued, and in August a mass was noted in the left iliac fossa. Although tuberculosis was suspected no tubercle bacilli could be found in her sputum. However, the baby failed to thrive and died at the age of eleven weeks. Post-mortem examination revealed widespread miliary tuberculosis. The liver was grossly involved, and the portal glands were caseous. Following these findings a laparotomy was performed on the patient, and tuberculous endometritis, salpingitis and peritonitis were found. The uterus, tubes and left ovary were removed. The patient was given a course of intramuscular streptomycin therapy, 0·6 gramme daily for two months. Investigation of her contacts revealed that her husband had active pulmonary tuberculosis, and her two children, aged four years and two years, had positive Mantoux test results but no obvious active tuberculosis. The patient was discharged from hospital in December, 1948. She was readmitted in March, 1949, when an X-ray examination showed miliary nodules of both lungs. She had a swelling of the right thumb, which eventually discharged pus in which acid-fast bacilli were found. She was discharged from hospital a week later, but was readmitted in October, 1949, because of hæmaturia and swelling of the right ankle. Full investigation failed to reveal tubercle bacilli in the urine, and her dysuria and ankle swelling subsided. Since then she had been well, and her chest X-ray appearance had become normal, but in January, 1951, she had developed a swelling over the upper third of her left tibia. An X-ray examination revealed a bone abscess. She was given a further course of streptomycin (60 grammes) and PAS, and the lump had since subsided, although the X-ray film still showed a cavity in the bone. She was now clinically well. The baby probably had congenital tuberculosis and had had no contact with the mother after birth. However, it had been in contact with the father for a week, so that post-natal infection could not be excluded.

Lung Abscess and Bronchiectasis.

Dr. Gould's second patient, a man, aged twenty-six years, had been admitted to hospital in December, 1950. His chest had been examined radiologically after a mild injury to the left side of his chest three weeks before. The examination showed a fracture of the left ninth rib, but also revealed a cavity two centimetres in diameter in the right upper zone, with some surrounding infiltration, and some linear opacities in the lower lobe of the left lung suggesting bronchiectasis. In the past nine years he had had two attacks of pneumonia, and his chest had been investigated in 1946. For three months he had had cough with yellow sputum and since the accident had coughed up a little blood. His fingers were grossly clubbed. In spite of the X-ray appearance numerous examinations and cultures of his sputum for tubercle bacilli yielded negative results. He was given 1,000,000 units of penicillin a day for three weeks, and at the end of that time the opacities in the upper zone of the right lung and lower zone of the left lung had almost completely resolved. A bronchogram showed cylindrical bronchiectasis of the basal segments of the lower lobe of the left lung. A bronchoscopic examination revealed no abnormality. It was felt that he had a lung abscess in the upper lobe of his right lung due to a long-standing left basal bronchiectasis, and that the fractured rib was incidental. The clubbing of his fingers had almost disappeared six weeks after his admission to hospital.

Carcinoma of the Lung with Cystic Changes in a Girl.

The next patient, a girl, aged fourteen years, had been first admitted to hospital in September, 1949. She complained of dyspnœa on exertion for three years. For two weeks she had had pleuritic pain in the left side of the chest and a non-productive cough. The chest X-ray film showed diffuse opacity throughout the left lung. That was regarded as due to pneumonia and responded to penicillin therapy. On the patient's discharge from hospital two weeks later her chest X-ray film showed slight clouding of the left lung base only. She was readmitted to hospital in August, 1950. For the past six months she had had pain in the left side of the chest. She had lost two stone in weight, and dyspnœa was now present at rest. There had been cough with some yellow sputum but no hæmoptysis. A chest X-ray examination showed some collapse of the left lung, and there was a uniform opacity except in the upper zone where cystic changes could be seen. A bronchogram showed a block of the left lower lobe bronchus with a smooth convex margin. Bronchoscopy showed a round tumour blocking the left lower lobe bronchus just below the left upper lobe orifice. A left pneumonectomy was performed in October, 1950, and the convalescence was uneventful. Section of the lung showed what was thought to be a bronchial adenoma, but on microscopic section this was found to be a definite infiltrating adenocarcinoma. The lung parenchyma of both lobes was almost entirely replaced by epithelialized cysts up to one centimetre in diameter. At the time of the meeting the patient was well.

Lung Cavity Probably following Hydatid Disease.

Dr. Gould's last patient, a man, aged seventy-six years, had been admitted to hospital in June, 1951, because of hæmatemesis. No cause for the hæmatemesis was found, but a chest X-ray examination showed a cavity in the lower lobe of the right lung, three centimetres in diameter, with some surrounding infiltration. Questioning revealed that the patient had had a hydatid cyst of his lung at the age of fourteen years. This he had coughed up. Apparently following this he had had a lung abscess and coughed up foul sputum for about twelve months. Since that time, apart from a mild cough and a slight amount of sputum, he had had no chest trouble whatever. His fingers were slightly clubbed. Sputum culture yielded negative results for

tubercle bacilli. In spite of negative results from Casoni and hydatid complement-fixation tests, it was thought that the cavity was the result of an old hydatid cyst.

Allergic Conditions.

DR. P. WARD FARMER showed three patients who suffered from seasonal hay fever, the symptoms starting in October and ending at Christmas in each case. Each reacted strongly to the common grass pollens, such as ryegrass, cocksfoot, Kentucky blue and Yorkshire fog. There were small reactions to plantain pollen. Testing was carried out by the scratch method with whole pollen. The technique of skin testing was demonstrated. Dr. Farmer discussed the relative merits of preseasonal and perennial treatment. He said that in preseasonal treatment injections with a pollen extract were started in June, and when pollens became plentiful in November the same dose was repeated weekly. In perennial treatment maintenance doses were given throughout the year, injections being given every fortnight when the maximum dose tolerated by the patient was reached. The method entailed fewer injections, but most patients preferred preseasonal treatment as a matter of convenience. Discussing the use of antihistamine drugs, Dr. Farmer said that most patients obtained some benefit from them, and they were also useful if an overdose of pollen extract was given. Milk should be avoided during the pollinating season as some pollen seemed to be secreted in cow's milk.

Dr. Farmer also showed a boy, aged seven years, who had suffered from eczema since the age of three months. Eczema symptoms had persisted, but now were waning. Symptoms of nasal allergy had been present during the last four years and had become worse in the last two months (August, September). Skin tests showed reactions to grass pollens and "house dust". Dr. Farmer thought that it would be worth while starting desensitization with a dust and pollen extract at the present time. It was explained that about 50% of subjects of infantile eczema were allergic to horse dander and egg-white at birth, but lost those sensitivities at about the age of five years and became sensitive to "house dust" and grass pollens and sometimes other allergens such as kapok and linseed meal.

Pathological Demonstration.

DR. J. F. FUNDER and DR. D. C. FORSTER presented a demonstration of pathological specimens. The exhibits included specimens of renal amyloidosis associated with rheumatic arthritis, primary carcinoma of the liver with intrahepatic and suprarenal metastases, neurilemmoma of the posterior cord of the brachial plexus, *Torula histolytica* meningoencephalitis and pulmonary infection, and specimens illustrating macroscopic types of carcinoma of the stomach.

The Diabetic Syndrome.

DR. JOHN STAWELL showed a series of patients illustrating some of the more uncommon examples of the diabetic syndrome.

Calcification of Pancreas.

Dr. Stawell's first patient, a man, aged forty-seven years, for twelve months had complained of colicky abdominal pain and backache. At no time was the pain very acute, but it was sufficiently intense to make him give up his work in a woodyard. Four months before being examined he had developed polydipsia and polyuria, and had started to lose weight. When first examined he had a moderate degree of diabetic acidosis. He was treated with insulin and diet. It was observed that his abdominal pain disappeared within twenty-four hours of his commencing treatment and never recurred. Investigation of the abdominal pain had revealed pronounced calcification of the pancreas. Further investigation of pancreatic function had revealed no abnormality.

Acromegaly.

Dr. Stawell's second patient was a woman, aged thirty-three years, suffering from acromegaly. She had been first examined four years before the time of the meeting, when she presented the typical signs and symptoms of acromegaly. Owing to severe headache and partial loss of vision in her left eye, she was operated on, and a chromophobe adenoma was removed from the pituitary gland by Dr. R. Hooper. Dr. Stawell showed a series of glucose tolerance curves prepared from the patient. The first, prepared before operation, was within normal limits, the second, prepared six months after operation when the patient presented with symptoms of thirst and polyuria, was definitely diabetic with a fasting blood sugar content of 0·23 gramme *per centum*. The patient had been treated with diet and insulin, the dose of which had gradually fallen. Finally, twelve months after the operation, insulin was no longer required, and at the present time the patient took no special diet, and the glucose tolerance curve was within normal limits.

In discussing this case, Dr. Stawell stated that it was most unusual, because the temporary diabetes might well have been caused by damage to the pituitary gland at the time of operation.

Acromegaly and Myxœdema.

Dr. Stawell's third patient was a man, aged fifty-three years, suffering from acromegaly and myxœdema. He had been originally stabilized on diet and protamine zinc insulin, 16 units daily. At that time his basal metabolic rate was −32%. On the exhibition of thyroid his basal metabolic rate had been brought up to −6%, and that necessitated increasing his insulin intake to protamine zinc insulin, 36 units daily, regular insulin, 20 units daily.

Dr. Stawell pointed out that acromegalics were usually severe diabetics, and that in the present case the severity had originally been modified by the state of hypothyroidism.

Thyrotoxicosis.

Dr. Stawell's fourth patient was a woman, aged twenty-five years, who had been admitted to hospital for treatment of thyrotoxicosis with methyl thiouracil. While in hospital she had developed thirst and polyuria. Her urine was found to contain sugar, and her fasting blood sugar content was 0·37 gramme *per centum*. She was treated with diet, protamine zinc insulin, 28 units per day, and regular insulin, 20 units per day. Nine months later thyroidectomy was performed, and within a month of operation the patient had ceased to take insulin. Investigation, however, revealed that she still had a mild diabetic glucose tolerance curve. During the next eight months, the diabetes gradually became worse, and insulin was again required. Some twelve months after the operation there was a further recurrence of the thyrotoxicosis, and, at the patient's request, a further operation was performed, with again a rapid fall of insulin requirements.

In discussing the case Dr. Stawell said that he considered that the patient had had a preexistent diabetic state, which was aggravated by the thyrotoxicosis, and that it was most probable that eventually she would require a permanent daily dose of insulin.

Possible Adrenal Basophilism.

Dr. Stawell's last patient was a woman, aged thirty-one years, who had presented with a six months' history of increasing weight, hypertrichosis and intermittent amenorrhœa. Two weeks before being examined she had developed polyuria, pruritus and thirst. Clinical examination revealed no definite abnormality, and X-ray examinations of the pituitary fossa and of the renal areas revealed no abnormality. The urinary ketosteroids were within normal limits. She had a diabetic glucose tolerance curve. Despite being considerably overweight she failed to respond to a reduction type of diet, became mildly acidotic, and required protamine zinc insulin, 32 units, and regular insulin, 28 units, for stabilization.

In commenting on this patient, Dr. Stawell said that the type of case was not uncommon and the symptoms and signs were not severe enough to warrant further investigation such as a laparotomy. Another interesting point noted was that it was extremely difficult to reduce the weight of patients of the type under discussion.

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

TO THE EDITOR OF "THE COLONIST", OCTOBER 3, 1837.[1]

[*The Colonist*, October 5, 1837.]

Sir,

Having seen a paragraph in last week's Colonist, in which my name is mixed up in no very courteous manner, arising no doubt from a total ignorance of the real facts of the case, and of the characters of the persons implicated, I beg to submit to your notice the following extracts from public documents, by which you will perceive the propriety of

[1] From the original in the Mitchell Library, Sydney.

exercising more caution in future, if you wish to maintain the character of your paper as an advocate for truth and justice.

I am, Sir,
Your obedient humble servant,
J. V. THOMSON,
Deputy Inspector General.

Memorandum addressed to Alexander McLeay, Esq., Colonial Secretary, by His Excellency the Governor:

March 26, 1836.

Inform Dr Bowman[1] that His Majesty's Government having resolved to establish and maintain a *superior* Medical Staff, for the purpose of supervising and controlling the Medical Establishments connected with the Military and Convict branches of the public service in this colony J. V. Thompson[2] Esq Deputy Inspector General of Hospitals has been appointed to the Superintendence of the Colonial and Military Hospitals in New South Wales.

Direct Dr Bowman to acquaint the several Colonial Medical Officers of the appointment and arrival of Deputy Inspector General Thompson, desiring them to pay attention to all such instructions as he shall think proper to give to any of them.

Instruct the Postmaster-General, that from and after the last day of the present month, the office of Inspector of Colonial Hospitals is to be considered as having ceased.

(Signed) R.B. (RICHARD BOURKE).

Extract from General Orders, Sydney, May 10, 1837:[3]

No. 124.

Adverting to that charges brought by Colonial Surgeon Mitchell against his superior officer, for many of which there appears no adequate foundation. His Excellency thinks it sufficient on this occasion to express his displeasure at so insubordinate and improper a proceeding, and to inform Surgeon Mitchell that if any well founded complaint of his conduct towards the Deputy Inspector General of Hospitals shall be again brought by that officer before His Excellency, it will be visited by a measure of much greater severity.

K. SNODGRASS,
Lieut-Colonel, Major Brigade.

To the Deputy Inspector General of Hospitals:[3]

Sydney.
September 26, 1837.

Sir,

I am directed by the Lieut-General Commanding to inform you, that having taken measures for ascertaining the correctness of the allegations contained in the charges brought by you against Colonial Surgeon Mitchell, His Excellency considers that officer to have *wilfully* and *deliberately* disobeyed the order of the Head of his Department, in refusing to attend punishments at Hyde Park Barracks and in omitting to sign the orderly book when required to do so. You will therefore inform Mr Mitchell that His Excellency has directed his name to be removed from the list of Colonial-Surgeons.

WILLIAM HUNTER,
Assistant Military Secretary.

Medical Societies.

MELBOURNE PÆDIATRIC SOCIETY.

A MEETING of the Melbourne Pædiatric Society was held at the Children's Hospital, Carlton, Victoria, on June 13, 1951. The meeting took the form of a series of clinical demonstrations by members of the medical staff of the hospital.

Sporadic Cretinism.

DR. A. M. HUTSON, in presenting a patient with sporadic cretinism, stated that he should make some apology for showing a patient with such a well-known entity to the Pædiatric Society. In justification, he stated that the condition was really quite a rarity, and it was over five years since he had seen such a baby in the out-patient department, so that there had been no opportunity to demonstrate a case to students. There must be many practitioners who, although familiar with the condition, had never actually seen a cretin. By contrast, of course, other conditions in which mental deficiency, particularly mongolism, presented were very commonly seen, and despite the clear differentiation between those conditions and cretinism, a surprising number of errors were made in children referred to the hospital.

The patient was a male child, aged thirteen months, brought to the hospital by his mother five weeks previously because of the sallow yellow colour of his skin, and because during the previous six months his rate of progress in weight and in general and mental development had been slow. He was the second child of normal parents; the first child was normal except for the presence of hypospadias. At birth he had weighed seven pounds and his length was 20 inches. He was jaundiced for several days a few days after birth.

During the child's first six months of life no definite abnormality was noted, but it was recalled that he had always been a quiet, placid and lazy baby and that the gain in weight was slow. The skin had always been sallow, and occasionally assumed a definitely yellow tint. Constipation had become evident at a few weeks of age and continued. An umbilical hernia was present. At the age of six months the mother took him to her doctor to seek advice concerning the hernia, and at that time no other abnormality was noted.

During the second six months of life the mother noted that the constipation was more severe, a bowel action occurring only every four or five days despite the use of laxatives and suppositories. The yellow colour in the skin became more apparent, and fluctuated considerably from time to time. The skin was always a little dry and on the face felt thickened, and the limbs were always cold. Hot water bottles were used even during warm weather. The facial appearance began to change, the baby developed a pale and wizened look, and the tongue was seen to be large and often protruding through the lips. He was slow in sitting up, not achieving this until the age of nine months, and even at thirteen months he showed little interest in standing or crawling. No teeth erupted, and the umbilical hernia remained large. The disposition of the baby was placid; he was easily managed and cried very little. He smiled a good deal, but often looked miserable; he laughed occasionally, but made no definite voice sounds. He did not display much vigour or curiosity and appeared quite content to sit or lie in his perambulator.

On examination of the child the striking features were the small stature and the pale, yellowish, wizened facies. The weight was 16 pounds and the height 26 inches (average normal figures were 22 pounds and 30 inches). The rectal temperature was 98° F., and the pulse rate 110 per minute. The lemon-yellow pallor was very conspicuous, giving an appearance closely resembling that of an adult with pernicious anæmia. The head was of normal size, the circumference being 18 inches, and the anterior fontanelle was open; but the face was small and wizened in appearance. The eyes were widely set, and there was a moderate degree of puffy swelling of the lower eyelids. The hair was fair, fine in texture and dry to the touch. The tongue was clean, large but not fissured, with the tip protruding through the lips. No teeth were present. The thyroid gland could not be felt. The skin of the body and limbs was pallid, slightly rough and cool to the touch. The hands were slightly short, with rather thick fingers. The heart and lungs were normal, the spleen and liver could not be felt, and an umbilical hernia was present. The expression was not completely dull, some alertness was apparent, but the general level of activity was low; the baby was content to lie quietly and was not easily disturbed. The hæmoglobin value was 12 grammes *per centum*, a surprisingly high value considering the evident pallor and the absence of a Wassermann reaction.

X-ray examination was made of the limbs, skull and spine. The films of the wrist showed that the stage of bone maturation was equivalent to that usually attained at birth. There were no epiphyseal centres present in the carpus or at the lower lid of the radius. All the films of the long bones showed a line or band of density at the metaphyseal ends suggesting the retardation or arrest of growth. The epiphyses were present for the lower ends of the femora and the upper ends of the tibiæ and had a degree of irregular mineralization giving a spotted appearance. Dr. Hutson commented that this appearace was more pronounced in older cretins and had been termed cretinoid epiphyseal dysgenesis. The films of the patient's skull showed retarda-

[1] Surgeon James Bowman, R.N., Inspector of Colonial Hospitals, 1828 to 1836.

[2] John Vaughan Thompson, Deputy Inspector-General of Hospitals, 1836 to 1844.

[3] From the original in the Mitchell Library, Sydney.

tion of maturation, with poorly developed diplöic markings and delay in closure of the sutures. Estimations of the basal metabolic rate and of the serum cholesterol content were not made.

In summary the presenting symptoms were lemon-yellow pallor, constipation, coldness and umbilical hernia, together with retardation of mental and physical growth. Inspection confirmed those, and disclosed a characteristic though not gross facies together with retardation of skeletal maturation.

Treatment was begun with Thyroid Extract, B.P., in a dose of one-quarter of a grain per day, increased after a week to one-half of a grain per day.

At the end of four weeks a considerable change was noted in both the general appearance and the degree of bodily activity. The mother stated that the constipation had been relieved after a few days and that now a bowel action was expected every day. The appetite had improved, but there was no gain in weight. The liveliness and activity of the child had increased; he now laughed and kicked his legs all day, would stand up and had begun to crawl. He had made attempts at vocalization and cried more vigorously. The pallor and yellowness had faded considerably, but were still recognizable. The hernia was less conspicuous.

X-ray examination after the first month of treatment showed that no epiphyseal centres had appeared in the carpus as yet; it was estimated that they should appear at the lower end of the radius, and in the capitate and hamate bones in another month or so. There had been, however, a pronounced spurt of growth, and the lines of density at the ends of the long bones had given place to new-bone formation to an approximate distance of 1·5 millimetres. That was well seen in the lower end of the ulna and in the femur.

Dr. Hutson said that an interesting finding in the case under discussion was the definitely yellowish discoloration of the skin, which was said to be due to an increased blood level of carotene. There had been no opportunity to confirm that; as a feature of cretinism it had been described in several accounts of the condition, but was not mentioned in some of the standard texts. The causation of the carotenæmia was unknown.

Dr. Hutson said that the initial response to treatment in a relatively mild cretin had been so satisfactory that an optimistic prognosis was possibly justified, despite the lateness of the diagnosis. The condition was definite enough, but even with the child at the age of thirteen months some observers were not confident that the diagnosis was correct, until the radiographic evidence was produced. Treatment would need to be continued throughout life, and the dose of thyroid increased when necessary; two to three grains per day were usually necessary in later childhood. It seemed probable that the physical growth would approximate to the normal, but the degree of mental retardation that would remain could not be estimated yet. It had been stated that even when cretins were treated adequately very early in life, the intelligence quotient rarely exceeded 90%. Dr. Hutson said that the child had been shown at the present early stage to give members of the Pædiatric Society the opportunity to observe the characteristic appearances before they were completely modified by the therapy.

DR. G. E. DOERY and DR. ROBERT SOUTHBY both commented on the fact that it was wise in babies with cretinism to commence therapy with thyroid extract in small doses. Some infants seemed peculiarly susceptible to thyroid therapy, and as there was no means of detecting which patients were sensitive, it was wiser to commence with a small dose and to increase this gradually until the maximum dose was reached. Dr. Southby further made a comment that the yellowish type of pallor was often very characteristic in some of the early cases of cretinism that he had encountered.

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

General Revision Course, 1952.

THE Post-Graduate Committee in Medicine in the University of Sydney announces that the annual general revision course will be held in Sydney for two weeks beginning April 28, 1952. The course, whilst of a general nature, has been specially designed to give emphasis to the diagnosis and treatment of cancer, and is largely taken up with seminars, panel discussions and periods set aside for general discussion, questions and films. The full programme will shortly be published. Dr. G. L. McDonald will be supervisor of the course, and fees for attendance will be as follows: full course, £8 8s.; mornings or afternoons only, £4 4s.; one week only, £4 4s. This includes morning and afternoon teas. Early application, enclosing remittance, should be made to the Course Secretary, the Post-Graduate Committee in Medicine, 131 Macquarie Street, Sydney. Telephones: BU 5238 and BW 7483. Telegraphic address: "Postgrad, Sydney."

Course in Occupational Medicine.

The Post-Graduate Committee in Medicine announces that in conjunction with the School of Public Health and Tropical Medicine, a course in occupational medicine will be conducted for two weeks, from May 26 to June 6, 1952, under the supervision of Dr. Gordon Smith, and will consist of a series of lecture-discussions at the School of Public Health and Tropical Medicine, and several factory excursions. The course will be suitable for general practitioners and others interested in occupational medicine. Fee for attendance will be £1 1s,, and medical practitioners who wish to attend the course should apply before April 28, 1952, to the Course Secretary, the Post-Graduate Committee in Medicine, 131 Macquarie Street, Sydney.

THE MELBOURNE PERMANENT POST-GRADUATE COMMITTEE.

Special Courses in Hæmatology Suitable for M.D. Part II and M.R.A.C.P.

A SPECIAL COURSE in hæmatology suitable for candidates for M.D. Part II and M.R.A.C.P. examinations will be held in February and March, 1952. The arrangements are as follows: February 26, introductory, Dr. J. Bolton, at the Royal Melbourne Hospital. February 29, "Leuchæmia", Dr. John McLean, at the Alfred Hospital. March 4, "Macrocytic Anæmias", Dr. J. Bolton, at the Royal Melbourne Hospital. March 7, "Bleeding Diseases", Dr. John McLean, at the Alfred Hospital. March 11, "Nutritional Anæmia", Dr. John Colebatch, at the Children's Hospital. March 14, "Hæmolytic Anæmias", Dr. J. Bolton, at the Royal Melbourne Hospital.

Special Course in Pædiatrics Suitable for M.D. Part II and M.R.A.C.P.

A course of clinical lecture-demonstrations in pædiatric disorders suitable for candidates for M.D. Part II and M.R.A.C.P. examinations, arranged by Dr. Mostyn L. Powell, will be conducted at the Children's Hospital, Carlton, on Tuesday and Friday afternoons as follows, commencing at 2 p.m.: March 18, "The Problems of Chronic Cough", Dr. Howard Williams. March 21, "Certain Endocrine Disorders in Childhood", Dr. Norman Wettenhall. March 25, "Nephritis in Childhood", Dr. Henry Sinn. March 28, "Gastro-Intestinal Disorders of Infancy and Childhood", Dr. Vernon Collins. April 1, "Meningitis in Childhood", Dr. Stanley Williams. April 4, "Acute Pulmonary Lesions in Childhood", Dr. Medwyn Hutson.

PUBLIC HEALTH AND INDUSTRIAL HEALTH.

THE Royal Institute of Public Health and Hygiene announces that the next bi-annual course of instruction for medical men and women for the certificate in public health, and for the diploma in industrial health (part I), will commence on Friday, March 21, 1952, and the following one on October 3, 1952. This course leads to courses for the diploma in public health, and for the diploma in industrial health (part II), respectively. (All courses may be taken either whole time or part time.) Prospectuses, enrolment forms and full details may be obtained from the secretary of the Royal Institute of Public Health and Hygiene, 28 Portland Place, London, W.1, England.

WALTER AND ELIZA HALL TRAVELLING FELLOWSHIP.

APPLICATIONS are invited from medical graduates for the Walter and Eliza Hall Travelling Fellowship. The value of the fellowship is £500 *per annum*. The period of tenure is two years abroad and one year in Sydney. Further details

can be obtained from the University of Sydney Calendar or from the Dean of the Faculty of Medicine in the University of Sydney.

The Royal Australasian College of Physicians.

ALLOCATION OF CORTISONE.

THE Royal Australasian College of Physicians wishes to remind medical practitioners that all applications for cortisone should now be sent direct to the Deputy Director, Commonwealth Department of Health, in each State. Applications should include the following details: (*a*) Name, age and occupation of patient. (*b*) Diagnosis. (*c*) Essential clinical data of each case; where applicable, these should include the following: (i) blood count, including hæmoglobin estimation and eosinophile cell count; (ii) erythrocyte sedimentation rate; (iii) X-ray examination of bones, joints and chest; (iv) presence of signs of cardiac and/or renal disease, tuberculosis, *diabetes mellitus* and syphilis; (v) electrocardiogram, serum potassium content, serum chloride content, serum protein content, estimation of carbon dioxide combining power and urinary steroid levels. (*d*) An estimate of the amount of the drug required and whether suspension or tablet form is preferred. (*e*) Details of where the treatment is to be carried out. (*f*) An undertaking to supply a full report of results of treatment in each particular case including details of the investigations listed above.

Applications will be considered for allocation of cortisone for the following diseases which, in the opinion of the executive committee of The Royal Australasian College of Physicians, may be suitably treated by cortisone: (*a*) Acute rheumatic fever, particularly with carditis. (*b*) Rheumatoid arthritis, of recent onset with reversible manifestations in the younger age and wage-earning group. (*c*) Still's disease. (*d*) Ankylosing spondylitis. (*e*) Psoriatic arthritis. (*f*) Acute disseminated *lupus erythematosus*. (*g*) *Periarteritis nodosa*. (*h*) Acute gout. (*i*) *Status asthmaticus*. (*j*) Acute drug reaction including serum sickness, severe. (*k*) Exfoliative dermatitis, severe. (*l*) Pemphigus, severe. (*m*) Scleroderma, (*n*) Dermatomyositis. (*o*) *Erythema nodosum*. (*p*) Addison's disease and other types of adrenal cortical insufficiency. (*q*) Nephrosis, including Ellis Type II nephritis. (*r*) Acute hæmolytic anæmia. (*s*) Burns. (*t*) Acute surgical shock. (*u*) Acute inflammatory eye diseases, including sympathetic ophthalmia. (*v*) Leuchæmia. (*w*) Sarcoidosis. (*x*) Multiple myeloma. (*y*) Ulcerative colitis. (*z*) Acute medical or surgical emergencies in which the use of cortisone could be a life-saving measure.

Obituary.

JOHN CADELL WINDEYER.

THE following appreciation of the late Professor John Cadell Windeyer has been forwarded by Sir George Wilson.

As I have always looked on the late John Cadell Windeyer as one of my greatest friends, I felt that I should like to add my appreciation to the remarks made on his life's work by Dr. Grace Cuthbert which appeared in the journal of January 19, 1952.

Jack, as he was to his friends, and I started our medical course in the same year. We were great friends from the start, and I recollect at least two short vacations that I spent at his mother's home at Raymond Terrace near Newcastle. We qualified at the same time, and after being house surgeons in Sydney we both went to England for postgraduate work. We lived together in London and worked at various hospitals, doing mostly gynæcology and obstetrics, but we had a three months' course in pathology with Professor Bullock at the London Hospital. Later we were in residence for five months at the Rotunda Hospital in Dublin and afterwards went to Vienna to work in the clinic of Professor Chrobak and Professor Kraner in the *Allgemeines Krankenhaus* for about six months. At the end of about two years we both returned to Australia. Windeyer started practice in Sydney, while I drifted to Adelaide, but we kept in

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED JANUARY 19, 1952.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	..	..	..	..	..	..	..	..	..
Amœbiasis	..	..	..	..	..	..	..	..	..
Ancylostomiasis	..	..	1(1)	..	..	..	..	..	1
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	..	..	..	..	..	..	..	..
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	..	..	..	..	..	..	..	..
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	..	2	..	..	..	3	..	..	5
Diphtheria	1	8(3)	2(1)	..	2	..	..	..	13
Dysentery (Bacillary)	..	2(2)	..	..	..	..	..	..	2
Encephalitis	..	..	..	..	..	..	..	..	..
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	2	..	..	..	..	..	..	2
Infective Hepatitis	..	..	..	..	2(1)	..	..	..	2
Lead Poisoning	..	..	..	..	..	..	..	..	..
Leprosy	..	..	1	..	4	..	..	..	5
Leptospirosis	..	..	2	..	..	..	..	..	2
Malaria	..	..	2(2)	..	..	..	..	..	2
Meningococcal Infection	2(1)	1(1)	..	..	..	..	..	..	3
Ophthalmia	..	..	..	..	..	..	..	..	..
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	8(2)	7(4)	8(4)	28(18)	..	..	..	..	51
Puerperal Fever	..	..	1	..	..	..	..	..	1
Rubella	..	11(3)	1(1)	..	3(2)	..	..	1	16
Salmonella Infection	..	..	..	..	..	..	..	..	..
Scarlet Fever	11(8)	8(5)	4(2)	2(2)	..	3	..	..	28
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	1	..	..	..	..	..	..	1
Trachoma	..	..	..	..	..	..	..	..	..
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	40(30)	33(28)	12(10)	12(11)	11(8)	3	1	1	113
Typhoid Fever	..	1(1)	..	..	..	..	..	..	1
Typhus (Flea-, Mite- and Tick-borne)	2(1)	..	1	..	..	..	..	..	3
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

touch with each other. When he was appointed professor of obstetrics I always spent a good many hours in his department when I visited Sydney, and as I was responsible for the teaching of obstetrics at the University of Adelaide, we always had various problems to discuss. We often referred to the difference in teaching of obstetrics in relation to what obtained when he and I did our practical obstetrics at the old Benevolent Asylum in Sydney.

I think I was one of the first to whom he sent a copy of his pamphlet on "Abdominal Palpation in Obstetrics", which marked a new era in this method of teaching. He was always keen on his teaching, and from my knowledge of some of his students, his thoroughness and wisdom in this respect were fully appreciated. His slogan was always "to try and make childbirth safer for the mother".

Windeyer, Marshall Allan of Melbourne and myself were the three representatives for our respective States for many years for the Royal College of Obstetricians and Gynæcologists, and this kept Jack and me in constant touch. We were both very averse to the suggestion made that gynæcology and obstetrics should be merged in the College of Surgeons of Australasia which had recently been started.

Dr. Grace Cuthbert has given a full résumé of Windeyer's activities during his professional life, and there is only one point that I would refer to, namely, the establishment of the earliest antenatal clinic. I had received permission from the board of management of the Adelaide Hospital to run a clinic for out-patients (antenatal patients) one day a week in 1910, and I ran this single-handed until I went abroad at the end of 1914. This clinic then lapsed, but was revived in 1920 and later transferred to the Queen Victoria Maternity Hospital, so I think that this clinic antedated Windeyer's clinic at the Royal Hospital for Women.

As the first professor of obstetrics Windeyer certainly made his mark as a pioneer teacher of obstetrics, and it was very fitting that he should have been appointed Emeritus Professor on his retirement from the chair of obstetrics.

Medical Prizes.

PRIZE FOR A MEDICO-SURGICAL FILM.

A PRIZE of 100,000 francs and several other prizes are offered by *La Presse médicale* for medico-surgical teaching films. The competition is open only to amateurs. The films must not have been published, nor must they have been subsidized or produced by any laboratory or firm.

The films will be judged on their teaching value as well as on their cinematographic quality. They may be sound films or silent, coloured or black and white, but must be of 16-millimetre size.

Intending candidates should apply to the "Secrétariat de La Presse Médicale, Librairie Masson, 120 Boulevard Saint-Germain, Paris (6e)", before March 1, 1952.

Corrigendum.

IN the issue of THE MEDICAL JOURNAL OF AUSTRALIA of January 26, 1952, at page 128, is published an amendment to the regulations under the *Police Offences (Amendment) Act*, 1908, as amended. This refers to the *Police Offences (Amendment) Act*, 1908, as amended, of New South Wales.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Klajnowajg, Zygmunt, registered in accordance with *Medical Practitioners Act*, 1938-1951, Section 17 (1) (c), 6 Palmer Street, Belmore.

Wells, Ernest Kenneth Bethune, L.R.C.P., L.R.C.S. (Edinburgh), L.R.F.P.S. (Glasgow), 1951, 29 Kingston Street, Haberfield.

Walsh, John Raymond Warn, M.B., B.S., 1951 (Univ. Sydney), St. George Hospital, Kogarah.

Howell, David John, M.B., B.S., 1945 (Univ. Sydney), 3 Margaret Street, Ryde.

Duncan, Jewel Shirley Esther, M.B., B.S., 1946 (Univ. Sydney), cnr. Mann and Etna Streets, Gosford, New South Wales.

Morrissey, Matthew John, M.B., B.S., 1951 (Univ. Sydney), Sydney Hospital, Sydney.

Diary for the Month.

FEB. 18.—Victorian Branch, B.M.A.: Finance Subcommittee.
FEB. 19.—New South Wales Branch, B.M.A.: Council Meeting.
FEB. 21.—Victorian Branch, B.M.A.: Executive Committee.
FEB. 22.—Queensland Branch, B.M.A.: Council Meeting.
FEB. 24.—Tasmanian Branch, B.M.A.: Council Meeting.
FEB. 25.—Federal Council of the B.M.A. in Australia—Hobart.
FEB. 26.—New South Wales Branch, B.M.A.: Ethics Committee, and Medical Politics Committee.
FEB. 27.—Victorian Branch, B.M.A.: Council Meeting.
FEB. 28.—South Australian Branch, B.M.A.: Scientific Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federal Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178 North Terrace, Adelaide): All Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such notification is received within one month.

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